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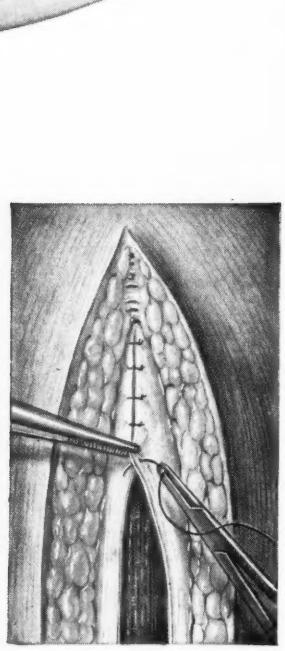

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SURGERY

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INEFFICIENT UTERINE ACTION

T. N. A. JEFFCOATE, M.D., F.R.C.S.E., F.R.C.O.G., K. BAKER, M.B.Ch.B., M.R.C.O.G., and R. H. MARTIN, M.B.Ch.B., M.R.C.O.G., Liverpool, England

THE safe and smooth delivery of a baby depends above all else on the efficient action of the uterine musculature. The expulsive effort of the upper segment must be intermittent and regular, and must be accompanied by relaxation and dilatation of the lower segment and cervix. This well known property of polarity can be represented diagrammatically (Fig. 1a). The more important of these two myometrial functions is the opening up of the lower segment and cervix to become the upper part of the birth canal. The less the resistance offered by these tissues the quicker and less distressing the labor. Differences in this resistance explain in large part why the multigravida has an easier delivery than her primigravid sister. The uterus pregnant for the first time is much less efficient at emptying itself than one which has had previous experience. In the primigravida the muscle is in higher tone but contracts more strongly and rhythmically, while the lower segment and cervix open easily.

The term "relaxation of the lower segment" is to some extent misleading because this part of the uterus is not entirely passive. It can contract and retract quite strongly as is evident during lower segment cesarean section. Moreover, if it were not so, every patient with placenta previa would die from postpartum hemorrhage. The inactivity of the lower uterus is relative to the strong activity of the upper, so in progressive labor there is, as Reynolds and others (1948) have happily described, a gradient of activity from fundus to cervix.

The contractions of the uterus in labor are also painful but the mechanism whereby the pain is produced remains uncertain. Some maintain that the pain is the result of progressive stretching of the sensitive lower pole of the uterus or of its adjacent ligaments and nerve ganglia. Others take the view that pain arises because the muscle is called upon to contract while it is in a state of ischemia, the ischemia being brought about by high muscle tone or retraction. Neither explanation accounts satisfactorily for all pain of uterine origin. For example, "after pains" can hardly be explained on the basis of stretching of the cervix or its ligaments. Indeed clinical observations suggest that both mechanisms can operate, but that each gives rise to a different sensation. Tension in the cervix and lower segment induces pain which is referred almost entirely to the lower part of the back. Hypogastric pain, on the other hand, seems to be closely related to activity of the upper segment. In normal labor, cervical resistance is minimal, and pain in the back is barely noticeable except sometimes at the inception of a "pain" and particularly during the final stages of dilatation. When, however, the lower segment and cervix offer unnatural resistance, or when they are in high tone,

From the Department of Obstetrics and Gynaecology, The University of Liverpool, England.

backache becomes a very prominent feature of labor. In general it appears that the less a woman experiences backache in labor, the more efficient is her uterus.

ABNORMAL UTERINE ACTION

There is much confusion over the different types of abnormal uterine behavior. Terms, such as "inertia," "inco-ordinate uterine action," "cervical dystocia," "constriction ring dystocia," are used indiscriminately to mean various conditions, while some writers evidently regard them as synonymous. Clarification of this situation is essential to rational treatment.

The following classification, a modification of one suggested previously (Jeffcoate 1949), attempts to reconcile most of the diverse clinical and experimental observations so far recorded, and is put forward tentatively as a working basis: (1) hypotonic inertia; (2) hypertonic states (a) hypertonic lower segment and internal os, (b) colicky uterus, (c) constriction ring dystocia; (3) asymmetrical uterine action; (4) cervical dystocia.

This grouping is probably more simple than is justified by clinical observation, and in some cases the uterus shows more than one disturbance. For example, hypertonus and asymmetrical action may occur together.

HYPOTONIC UTERINE INERTIA

In this condition relaxation of the lower uterus is adequate, but the expulsive effort is weak or infrequent or both. The pattern of activity is normal but the total is not enough for good progress of labor (Fig. 1c). According to Caldeyro, Alvarez, and Reynolds (1950) the intensity of contractions is not enough to raise the intrauterine pressure above 24 milligrams of mercury. The feeble activity and low tone of the uterine muscle means that the patient experiences little, if any, pain, and except for other signs such as some dilatation of the cervix, a show, or rupture of the membranes, it may be difficult to say that labor has commenced. The force exerted on the fetus and its placenta is small so molding and caput formation are usually limited, and there is little chance of intrauterine asphyxia. Labor may continue for days without involving great risk to either mother or fetus. This is true inertia; it can arise at any time in labor and it may precede or follow a period of normal uterine action.

HYPERTONIC STATES

Types. Hypertonic lower segment and cervix.—Here the second function of the uterus is disturbed: namely, the lower segment offers unnatural resistance to dilatation. Coincident with this, and obeying the laws of polarity, the action of the upper segment may be weak or absent (Fig. 1e). It can therefore be confused with hypotonic inertia. The essential difference is that, whereas in the latter the patient has no pain, hypertonus or any form of resistance in the lower segment and cervix leads to severe low backache. This disorder is sometimes called "reversed polarity," a phenomenon which has been seen in monkeys and induced in these animals by stimulation of the hypogastric sympathetic nerves.

Resistance in the lower part of the uterus occasionally manifests itself more or less entirely at the level of the internal os (Fig. 1i). The presenting part is then held high and the lower cervix is not exposed to the normal dilating mechanism, so it remains thick with little sign of effacement. There is no need to classify this condition separately, although it is not unreasonable to regard it as a form of constriction ring dystocia or to term it achalasia of the cervix. It should not, however, be called cervical dystocia, a term best reserved to describe labor mechanically obstructed by passive resistance at the level of the external os (Fig. 1j).

Colicky uterus.—The "colicky" uterus is characterized by strong but purposeless activity, normal polarity being absent (Fig. 1d). All parts of the uterus contract more or less equally, and in a colicky fashion; there is no gradient, so dilatation of the cervix and lower segment fails to take place or only does so slowly and with difficulty. The patient suffers intense and frequent abdominal cramps of a constant and high frequency, in contradistinction to the gradually increasing frequency of normal first stage pains. The uterus ultimately becomes tender and the patient resists examination.

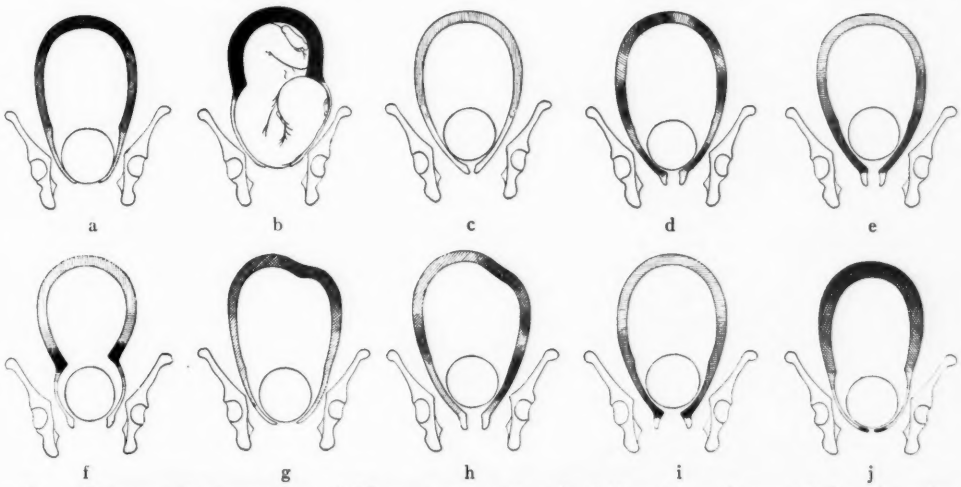

Fig. 1. Diagrammatic representation of different types of uterine activity, the depth of shading being indicative of the amount of the tone and activity in any one part of the organ. a, Normal action with fundal dominance. b, Normal pattern of action pathologically increased by obstructed labor. Exaggerated retraction ring (Bandl). c, Normal pattern of action but very weak. Hypotonic or true inertia. d, Colicky uterus. Irregular and generalized contractions. No gradient of activity. Cervix and lower segment not "taken up." e, Hypertonic lower segment. Reversed polarity. f, Constriction ring dystocia, a possible end result of (d) or (e). g, Asymmetrical uterine action, the pattern otherwise normal. This is probably of little practical significance. h, Asymmetrical and "colicky" uterine action. i, Hypertonicity localized to internal os, an excessive degree of (e). May also be called "achalasia" of the cervix. j, Cervical dystocia. A normal, or slightly exaggerated normal, pattern of activity with fundal dominance, effacement of the cervix, and engagement of the presenting part. The dark shading in the tip of the external os in this diagram represents passive resistance and not hypertonicity.

Constriction ring dystocia.—The formation of a constriction ring, or contraction ring, is to be regarded as an extension, or an end result, of either reversed polarity or of a colicky uterus. In it the condition has progressed so far that an annular area of the uterus becomes tonically and persistently contracted, and may become so tight as to grip the fetus and prevent its birth (Fig. 1f). The symptoms are exactly the same as those of the two other types of hypertonic incoordinate activity, and the diagnosis can therefore be difficult. The appearance of ring formation on abdominal examination can be deceptive and the diagnosis should only be made if the ring is felt on intrauterine palpation. Even then the contraction induced when a hand is inserted into the uterus of a partially anesthetized patient, or even the normal retraction ring, can be mistaken for a constriction ring. This is particularly true since the commonest site for the stricture is at the junction of the upper and lower segments. It can, however, occur in any part of the muscular uterus. The difference between a constriction ring and an exaggerated retraction ring (Bandl's ring), which is seen in obstructed labor, can also be illustrated diagrammatically (Fig. 1b). The two are essentially different in that Bandl's ring is a manifestation of efficient uterine action—it can only arise when polarity is normal. In neglected cases the ultimate outcome is rupture of the exceptionally thin lower segment. On the other hand, normal polarity is absent in constriction ring dystocia, the lower segment is not excessively thinned and spontaneous rupture *never* takes place.

Reversible constriction rings can be induced by clumsy intrauterine maneuvers carried out without deep anesthesia, and by the administration of oxytocics to a uterus already hypertonic and inco-ordinate. When, however, they arise spontaneously as the end result of hypertonic uterine action, it has been suggested that they represent a cramp

resulting from muscle fatigue. Most rings are reversible in that they relax under the influence of deep anesthesia or death. Very few authentic cases of irreversible ring formation have been described.

Clinical features. These three types of inco-ordinate uterine action have certain features in common. All are characterized by a state of hypertonus in some part of the uterus and by a persistently raised intrauterine pressure. The pains of labor are in some way associated with intrauterine pressure, and in this case the resting pressure approaches the critical level for pain production which is said to be about 25 millimeters of mercury. The patient tends therefore to feel discomfort—and especially backache—in between contractions, with exacerbations as the uterus contracts. Moreover, unlike the normal state of affairs, the onset of pain precedes the onset of a palpable contraction, and it continues even when the contraction appears to have died away (Fig. 2). It is the persistently raised intrauterine pressure which, by interfering with the placental circulation, threatens the fetus with asphyxia, and this danger is present even when the membranes are unruptured.

The cervix in these conditions not only dilates slowly, but tends to remain thick and incompletely effaced. The fetal head is not driven firmly on to it, so it often becomes edematous as well.

The overall clinical picture is one of prolonged labor and slow dilatation of the cervix despite uterine contractions which are in many cases strong. The contractions may be frequent or may be irregular in strength and periodicity, but the pains are unusually distressing and the patient is conscious that no progress is being made so she tends to become anxious and to lose morale. Sometimes, after a long period of first stage pains, the patient begins to bear down and the attendants are misled into thinking that the cervix is fully dilated. This false bearing down sensation, which is of great practical importance, appears to arise from a spasm in the rectum and pelvic colon. It is in fact a desire to evacuate the bowel rather than the vagina. It may well be evidence of a disturbed sympathetic nervous system, which so many regard as the cause of inco-ordinate uterine action. There are other evidences of this, too, in that the descending colon, and sometimes the stomach, become distended and bulge so much that they partly overlap the front of the uterus, causing or suggesting obliquity of the uterus. Again the patient finds it difficult to empty the bladder. It is said that a full bladder causes "inertia," but is this true? It may be the result rather than the cause. Or again, retention and faulty uterine action might both be parallel manifestations of an underlying autonomic nervous upset.

Vomiting is common and this, together with a failure to take nourishment, leads to dehydration. Ketosis and acetonuria appear sooner or later, and exhaustion with pyrexia and tachycardia set in.

ASYMMETRICAL UTERINE ACTION

Although of far less importance than co-ordination of the upper and lower segments of the uterus, synchronous activity of the embryological halves of the uterus is a function of normal uterine behavior. The part of the uterus developed from each müllerian duct has its own nerve supply, albeit with a good deal of intercommunication. Contraction waves normally arise independently, but more or less simultaneously, in each cornu and then meet to form one. When the uterus is bicornuate each horn can act independently, and even when it is apparently normally developed there may be differences in the activity of each half. However, if the contraction pattern is otherwise normal (Fig. 1g), this does not materially impede the progress of labor. Asynchronous activity can also occur in association with disturbed polarity, and it is this possibility which probably accounts for the one sided pain which many have described as a feature of a colicky uterus or constriction ring dystocia (Fig. 1h).

However, obvious anomalies of müllerian duct fusion are not a numerically important cause of serious uterine dysfunction in labor. When they are present the action of the uterus is to some extent dependent on the type of malformation. If the two halves of the uterus

are completely separate (uterus didelphys, uterus unicornis) each half may function well. The most trouble arises when the fusion is incomplete as in uterus bicornis unicollis or uterus subseptus.

CERVICAL DYSTOCIA

This is a condition in which uterine action is well co-ordinated, expulsion of the fetus being prevented by a mechanical obstruction at the external os (Fig. 1j). The lowermost part of the cervix, unlike the remainder, consists of fibrous or collagenous tissue rather than muscle, and this normally undergoes extreme softening—almost liquefaction—in late pregnancy (Hughesdon, 1951). If for some reason the softening process is defective the external os fails to dilate. An efficient uterine muscle then brings about normal effacement of the cervix, but delivery is prevented by a thin hard ring of external os. The essentially normal behavior of the uterine muscle, including that of the lower segment and upper cervix, is evidenced by the fact that the presenting part is always well applied to the cervix and is driven low into the pelvis, indeed there are cases recorded in which the fetal head has been extruded through the vulva still covered by an undilated intact cervix. Once the ring is divided, or if it tears, a rapid and easy delivery gives further proof of a normal contraction pattern.

Cervical dystocia indeed is a condition of obstructed labor and if it occurs in a multigravida—as it sometimes does when the cervix is fibrosed as a result of injury or disease—the typical uterine response to obstruction, excessive retraction going on to spontaneous rupture, is seen. Primary cervical dystocia, however, occurs only in primigravidas and, as always in obstructed labor in such patients, the uterine response is not one of increased activity and rupture, but rather inertia. The diagnosis of cervical dystocia then depends on the character of the cervix and its close application to a presenting part driven low in the pelvis (Fig. 1j). The patient experiences backache because of the tension which is present in the cervix and this pain becomes more intense if any attempt is made to stretch the external os.

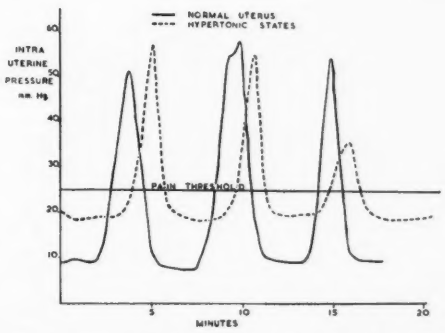

Fig. 2. Diagrammatic representation of pain threshold in relation to intrauterine pressure. In normal uterine action the pressure falls to less than 10 millimeters of mercury, well below the critical level, in the intervals between contractions. In hypertonic states of the uterus the resting pressure is high so pain is felt continuously or at the very inception of a contraction.

The rare condition of conglutination of the external os is to be regarded as a form of cervical dystocia.

A minor degree of cervical dystocia is not uncommonly seen in primigravidas early in labor, when the undilated external os of a cervix fully effaced becomes displaced upward and behind the presenting part. This is a source of error in assessing the degree of dilatation, especially on rectal examination. Less commonly the displacement occurs in a forward direction. The condition is sometimes called sacculation of the cervix, and as a rule it undergoes spontaneous cure. It can be accounted for on the basis of irregular and unequal softening of the cervical fibrous tissue so that one lip stretches more easily than the other. A few cases of this kind have been described in which the thin cervix has presented at the vulva and has been mistaken for the bag of membranes and deliberately punctured. One such case is illustrated in Figures 3 and 4 and is reported by kind permission of Mr. C. M. Marshall.

The patient attended hospital on account of uterovaginal prolapse, and in addition was found to have a fistula running from the posterior aspect of the vaginal cervix into the cervical canal. Her doctor then described how during the first labor there was some delay. He found what he regarded as a rather

Fig. 3. Case of fistula between upper cervical canal and posterior aspect of cervix. This probably arose as the result of posterior sacculation of the cervix in a case of cervical dystocia and two deliveries took place through it. Note the nulliparous appearance of the external os.

thick bag of forewaters presenting at the vulva and punctured these. Whereupon the rest of the delivery was easy. The second confinement was easy and unassisted, delivery having presumably again taken place through the artificial opening on the posterior aspect of the cervix. The illustration shows that the external os retains nulliparous characteristics.

Another interesting complication of cervical dystocia is spontaneous annular detachment of the cervix, and this condition is not so rare as is suggested by the fact that only about 60 cases have been recorded in the literature. We have personal knowledge of 4 further cases, which are described in a separate communication (Jeffcoate and Lister, 1952). Sloughing of a localized area of the cervix can arise under all manner of circumstances and is not of importance in this context, but annular detachment is of considerable significance so far as cervical dystocia is concerned. It is an injury which is primarily determined by prolonged arrest of the circulation in the dependent part of the cervix, this disturbance being the result of pressure which must be applied *equally* at all points of a ring of cervix, a circumstance most unlikely to occur as a result of disproportion as is sometimes suggested. The most probable mechanism for the equally distributed pressure is that the presenting part presses down against a usually resistant cervix. Indeed it is the cervix itself which supplies the counterpressure. Thinning of the cervix may also play a part in reducing its blood supply. Such pressure or tension leads to congestion, intravascular thrombosis, extravasation of blood, and devitalization of all that part of the cervix which lies below it and which is supplied by descending blood vessels. It is often assumed that the detachment of the cervix is due to sloughing alone. This is unlikely, however, because it takes place too soon after the circulatory arrest. For this and other reasons it is necessary to invoke a mechanical factor as well, and this in most cases is the retracting uterus pulling against the resistant external os. The cervix then tears around its most devitalized area at the level of the pressure ring. The most likely line of separation is the cervicovaginal junction. The level varies with the degree of dilatation of the cervix, and we cannot confirm that it marks the junction of the fibrous and muscular parts of the cervix as suggested by Schwarz and Woolf (1948).

TABLE I

Hospital	No. of cases all types	No. of consecutive deliveries analyzed	Incidence, per cent
M.R.H. 1946–1951 (5 year period) (unselected material)	105	13,917	0.8
L.M.H. 1948–1951 (3½ year period) (selected material)	110	8,799	1.4

In order to obtain sufficient force the uterus must be acting efficiently, and the fetal head must be well applied to the cervix which must be taken up. The fact that the cervix was thin and completely effaced is mentioned in most recorded cases of annular detachment. The condition, therefore, is never seen in inco-ordinate uterine action or inertia, and can only occur in cervical dystocia which may be either idiopathic or secondary to scarring or disease. The presence of organic resistance in the cervix, and of normal myometrial function above, is also evidenced by the fact that once the cervix becomes detached the remainder of the delivery is easy. Indeed this accident, although not common,

is an important link in the chain of evidence in support of the classification given of abnormal uterine action.

INCIDENCE OF INEFFICIENT UTERINE ACTION

Overall incidence. The duration and character of labor vary widely within normal limits, all grades of efficiency in uterine action are seen and the boundary between normal and abnormal is so vague that it cannot be defined except in terms of arbitrary and unsatisfactory figures of time. Nevertheless, it would be generally accepted that minor degrees of disturbance of uterine action, of the types already stated, are common—how common depends as much on the acuity as on the personal opinion of the observer. Minor disturbances, however, although trying for the patient and the attendant, are not serious and are either amenable to treatment or resolve themselves given a little time and patience.

It is the more severe types of disorderly uterine action, those in which labor continues for days, in which the cervix only becomes 2 or 3 fingers dilated in 48 to 72 hours, and in which the lives of the mother and fetus are threatened, which give rise to the serious clinical problems. And these are not so common as is sometimes suggested. In the practice of two large maternity hospitals in Liverpool the overall incidence of *all* types of severe uterine disorder in labor lies between 0.8 per cent and 1.5 per cent (Table I). If the hospital admissions are unselected, as they were at the Mill Road Maternity Hospital (M.R.H.) at the time in question, serious uterine inefficiency is not seen in more than 1.0 per cent of cases. The Liverpool Maternity Hospital (L. M.H.), however, drains a wide area and selects its admissions to include abnormal, or potentially abnormal, cases at the expense of the normal.

If these figures are compared with those published in the literature, a disparity will be noted. Thus some authorities say that cervical dystocia alone occurs in 1 per cent of cases, others that constriction rings are present in over 1 per cent of labors (Table II).

These differences are almost certainly accounted for by different concepts of uterine dysfunction, in other words some writers regard all cases as constriction ring dystocia, others as cervical dystocia. In our experience *proved* constriction ring dystocia does not occur more often than in 0.1 to 0.2 per cent of unselected deliveries, and the incidence of cervical dystocia in severe form is similar.

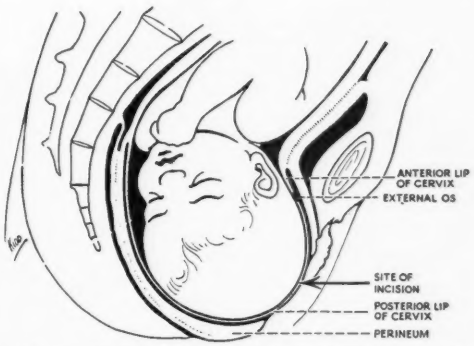

Fig. 4. Diagram illustrating the situation leading to the cervical fistula shown in Figure 3.

Those writers who report a rather high overall incidence of disorderly uterine action are including less severe types of case.

The 277 cases reported here comprise the two hospital series mentioned together with a smaller group from private practice (selected material). They include only those in which careful observation at the time, and retrospective analysis, put the diagnosis beyond all reasonable doubt. Spurious labor and minor departures from the normal with slight lengthening of labor are excluded, as are all cases in which serious disproportion was also

TABLE II

	All types per cent	Primary inertia, per cent	Cervical dystocia, per cent	Constriction ring dystocia, per cent
Odell and others (1945)	2.7			
Sackett (1941)			1	
Johnson (1946)				1.3
Rucker (1946)				1.7
Mills (1949)				1.5
Arthur (1949)			1.5	
MacRae (1949)		3.2		
Jeffcoate (present series)	0.8–1.4		0.1–0.2	0.1–0.2

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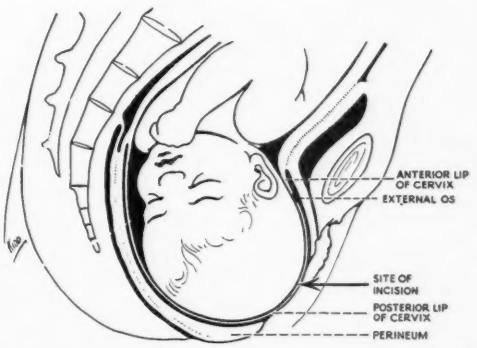

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TABLE II

	All types per cent	Primary inertia, per cent	Cervical dystocia, per cent	Constriction ring dystocia, per cent
Odell and others (1945)	2.7			
Sackett (1941)			1	
Johnson (1946)				1.3
Rucker (1946)				1.7
Mills (1949)				1.5
Arthur (1949)			1.5	
MacRae (1949)		3.2		
Jeffcoate (present series)	0.8–1.4		0.1–0.2	0.1–0.2

TABLE III.—DURATION OF LABOR

Duration hours	Private series No. cases	M.R.Hospital 1946 to 1951 No. cases	L.M.Hospital 1948 to 1951 No. cases	Total
24–35	13 (6)	15 (9)	19 (19)	47 (34)
36–47	6 (4)	38 (18)	19 (10)	63 (32)
48–59	15 (4)	24 (9)	40 (21)	79 (34)
60–71	8 (4)	16 (8)	12 (6)	36 (18)
72–83	9 (2)	8 (2)	13 (7)	30 (11)
84–95	2 (0)		5 (2)	7 (2)
96–107	6 (1)	3 (1)	1 (1)	10 (3)
108–119	1 (0)		1 (1)	2 (1)
120+	2 (2)	1 (1)		3 (3)
Totals	62 (23)	105 (48)	110 (67)	277 (138)

138 cases (in brackets) delivered by cesarean section before completion of first stage.
Average duration of incomplete first stage = 52 hours.
139 cases delivered vaginally.
Average duration of first and second stages = 55 hours.

TABLE IV.—PARITY

Age, years	Private series		M. R. Hospital series		L. M. Hospital series	
	Primigravidas No.	Multigravidas No.	Primigravidas No.	Multigravidas No.	Primigravidas No.	Multigravidas No.
Less than 20			8	1+1*		
20–24	7		37 (2)		25 (2)	1
25–29	17	1†	32	1*	30 (2)	1*
30–34	21 (1)	1	19 (2)	1*	29 (1)	2*
35–39	9 (1)	3	4 (1)		17 (1)	
40–44	2	1	1		4	1
45†						
Totals	56	6	101	4	105	5

*Previously delivered by C.S.
†This patient had a uterus didelphys and the other pregnancy had been delivered from the opposite horn.
( ) Figures in brackets indicate number of patients labelled primigravidas who had one or more early abortions.

present. No case less than 24 hours in labor is accepted, and a high proportion of those less than 36 hours in labor had made such little progress that they were delivered by cesarean section with the cervix only partially dilated. One hundred and thirty-nine cases were delivered vaginally and in these the average duration of the first and second stages of labor was 55 hours. One hundred and thirty-eight cases were delivered by cesarean section before full dilatation of the cervix, and in these the average duration of the incomplete first stage was 52 hours (See Table III).

Age and parity. Inefficient uterine action is almost entirely a disease of primigravidas, and it is rare to see it in multigravidas (see Table IV). The only multigravidas who suffer inefficient uterine action in a severe form are those who have never had their cervices fully dilated, having been previously delivered by cesarean section, and a few who develop either a colicky uterus or true inertia.

Although inco-ordinate uterine action is reputed to be more common in elderly primigravidas, especially those of low fertility, the effect of age is often exaggerated. It is easy to get a wrong impression from casual clinical observations, as can be seen by a comparison of the age incidence of faulty uterine action amongst primigravidas with the age of incidence of first childbearing in the same community (See Table V).

TABLE V.—AGE INCIDENCE (PRIMIGRAVIDAS ONLY)

Age, years	Private Series				M. R. Hospital				L. M. Hospital			
	500 consec. deliveries		Faulty uterine action (56 cases)		1,000 consec. deliveries		Faulty uterine action (101 cases)		1,000 consec. deliveries		Faulty uterine action (105 cases)	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Less than 20	5	1			131	13	8	8	58	6		
20–24	70	14	7	13	493	49	37	37	414	41	25	24
25–29	181	36	17	30	287	29	32	32	320	32	30	29
30–34	139	28	21	38	57	6	19	19	133	13	29	28
35–39	87	17	9	16	28	3	4	4	54	5	17	16
40–44	18	4	2	4	4		1		20	2	4	4
45+									1			
Average age, years	29.9		30.2		24.2		25.4		24.1		29.4	

These figures suggest that although elderly primigravidity tends to increase the chance of faulty uterine action, its effect is only slight. They go to confirm the findings of Rucker (1946) although he called all his cases constriction ring dystocia.

Rupture of the membranes. The time of rupture of the membranes does not appear to influence directly the incidence of faulty uterine action. In the cases under review the trouble occurred before rupture of the membranes in 169 cases, after premature rupture of the membranes in 71 cases, while information on this point is lacking in the remaining 37 cases.

In established hypertonic states, rupture of the membranes, either spontaneous or artificial, did not significantly improve uterine behavior. There were no cases of hydramnios in this series, and there were only 2 cases of multiple pregnancy. Overdistention of the uterus in fact did not appear to play any significant part in the incidence of inefficient uterine action.

Mechanical errors in relation of the fetus to the birth canal. Uterine inertia of any type is no more likely with breech than with vertex presentations, but nearly all observers note a significant association between it and posterior position of the occiput or transverse arrest of the head. Some obstetricians even recognize a "posterior" labor. It is impossible to say how these malpositions act, but it is generally postulated that anything which interferes with close and even application of the head to the lower segment in the first stage, or to the lower vagina and perineum in the second stage, reflexly interferes with uterine contractions. It has been argued that the failure of the head to rotate favorably is the result rather than the cause of uterine inefficiency, but clinical observations suggest that the malposition generally precedes the uterine disturbance, and that when it is corrected good uterine contractions often return. So it comes about that a posterior position of the occiput can frequently be diagnosed from the character of the pains, and women with obstetrical knowledge can sometimes during their own confinement say when the occiput is posterior and when it rotates to the front. The relatively sudden failure of uterine action at the end of the first, or early in second, stage of labor, is almost pathognomonic of posterior rotation of the occiput.

TABLE VI

	No. cases
Cephalic presentation	
Anterior position of vertex	83
Oblique or direct occipitoposterior position	87
Transverse position of vertex	93
Position not recorded	6
Breech presentation	8
Total	277

Ideas on the association between malposition of the occiput and inefficient uterine action, however, were formulated at a time when the mechanism of labor was misunderstood. It was not then recognized that the fetal head usually enters the brim with its sagittal diameter lying transversely. Unless the head is low in the pelvis, therefore, an occipitolateral position may be normal. Moreover, statistics dealing with the association do not make it clear whether the posterior or transverse position of the occiput was present at the time the disorderly action of the myometrium commenced, or whether they were only present at the time of delivery. Indeed, it is extremely difficult, if not impossible, to produce significant figures. Those in Table VI indicate the position of the fetus at the time when the uterine disorder was at its height, and in the majority of cases the same position was still present at the time delivery was effected.

Apart from the position of the presenting part, the question of its engagement is also important. Indeed one of the commonest and most important factors in inefficient uterine action is cephalopelvic disproportion. The primigravid uterus is inhibited in the presence of mechanical obstruction, so in cases of disorderly myometrial function it will often be found that either the pelvis is a little smaller than was expected, or that the fetus is unusually large. Indeed it is extremely difficult to analyze any case of prolonged labor in terms of faults in the passages, the powers and the passenger. More than one factor is often present and it can become a matter of opinion as to which is primary and the most important.

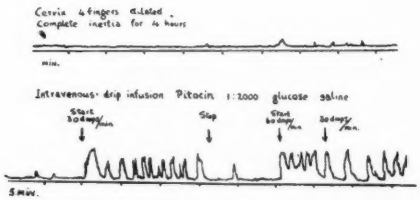

Fig. 5. Intravenous oxytocin drip therapy in hypotonic inertia. Tocograph before and after treatment showing immediate effect of starting and stopping infusion. When the speed of the drip was increased to 60 drops per minute contractions became strong and frequent and the base line was raised suggesting increase in tone. Normal behavior restored by reducing rate of flow. A. O'B., para 3, aged 33 years. Labor induced by artificial rupture of the membranes 2 weeks after estimated date of confinement; intermittent labor for 4 days.

Even when disproportion and malposition are excluded a high presenting part, and sometimes an onset of labor delayed beyond term, are often found in association with inefficient uterine action. The question then arises as to whether failure of engagement is the result, rather than the cause, of inco-ordinate muscular action. In other words, is the presenting part held high by hypertonicity of the lower segment present even before onset of labor?

In the same way, false labor can be regarded as a form of inco-ordinate uterine action. It occurs in two forms: (a) severe and constant backache, which is indicative of unusual resistance on the part of the lower segment and cervix to the "taking up" process which precedes labor; (b) strong and frequent muscle cramps without dilatation of the cervix; the action of the uterus is then similar to that described under the heading of "colicky uterus."

MANAGEMENT AND TREATMENT OF INEFFICIENT UTERINE ACTION

Diagnosis. The most important single step in the management of inefficient uterine action is a meticulous examination to find its cause and, above all, to exclude the presence of disproportion and malposition of the fetus. No matter how much interference there may have been previously, no decision as to treatment should be made without first carrying out vaginal examination, and, if facilities are available, intranatal roentgenography.

If there is no evidence of serious disproportion it is next necessary to determine the type of uterine disorder, and until a convenient and reliable method of recording myometrial activity is available, this can only be done by careful analysis of symptoms and signs. Four questions arise: (1) Is the labor real or spurious? (2) Is the uterus hypotonic and truly inert? (3) Is the uterus hypertonic and its action inco-ordinate? (4) Is uterine polarity normal with labor obstructed by passive resistance at the external os?

The essential points in the differential diagnosis of the last three have already been covered. If there is doubt about the answer to the first question, and there often is, a therapeutic test with morphia or other sedatives in adequate dosage should be applied. If such drugs allay uterine action completely, it is fairly safe to assume that real labor has not yet commenced.

General. The general management of prolonged labor is well known, but it deserves emphasis because it is probably more important than any specific remedy designed to affect the uterus. Maintenance of physical well being and of morale, and relief of pain, at least allow the patient to tolerate more labor thus offering more opportunity for spontaneous cure of the uterine disorder. The appearance of acetonuria always calls for intravenous drip infusion of 5 per cent glucose solution. Indeed continuous intravenous medication is one of the most significant recent advances in treatment. In addition to its general effects, glucose often appears to improve uterine action, and this fact offers some support to those who believe that spasm of the uterus, even a constriction ring, is the result of muscle exhaustion. Moreover, the infusion can be made the means of safe and controlled administration of oxytocics, analgesics, antispasmodics, and chemotherapeutic agents.

Artificial rupture of the membranes. Artificial rupture of the membranes is a simple treatment which may be effective in cases of true inertia. It is never of value, however, in hypertonic conditions.

Oxytocin. Posterior pituitary extract was first used in obstetrical practice by Blair-Bell (1909). Its abuse, however, soon brought it

into general disfavor except for the treatment of postpartum hemorrhage. The last 5 or 6 years have seen it reintroduced for the treatment of inertia, but it is given in more physiological dosage, administered by intravenous drip of solutions varying in strength from 1:1,000 to 1:10,000. This method was first suggested by Page (1943) and has been the subject of clinical reports by Theobald and colleagues (1948), Hellman and colleagues (1950), and others. We have used it in carefully selected cases since 1947, the strength of the solution being 5 units oxytocin in 1 liter of 5 per cent glucose solution. The method is safe because it gives the attendant almost complete control over the uterus, whose action increases or decreases within seconds or minutes of turning the stopcock (Fig. 5).

The value of oxytocin in faulty uterine action is, however, strictly limited. Its effect on any uterus is always to increase whatever type of action has been previously present. It does not alter the behavior pattern, so that if the uterus is already hypertonic and inco-ordinate, it makes matters worse (Fig. 7) and in such circumstances can cause a constriction ring. It should therefore only be employed when the uterus is hypotonic and truly inert but possessing normal polarity. Its effect then is dramatic and pleasing, but again there is need to add a note of caution. If the dose is too high, it may produce a rise in tone in the uterus and cause such violent action as to imperil the fetus (Fig. 5). Moreover, if the administration is continued for a long time it will often be found that the uterus becomes refractory, and it then fails to respond to further treatment (Fig. 6).

In cervical dystocia contractions are increased as might be expected, but dilatation of the rigid cervix is not materially influenced. Since the condition is one of obstructed labor this treatment is irrational and potentially dangerous. It might at least cause severe laceration of the cervix.

Analgesics and antispasmodics. The medical treatment of inco-ordinate uterine action consists in relieving pain, exhaustion and anxiety by analgesics and hypnotics, and in attempting to reduce uterine spasm and hypertonus by antispasmodics of one sort or another. The value of morphia, combined if need be with hyoscine or barbiturates, requires no emphasis. Nothing better has yet been produced. Pethidine, however, has perhaps a greater margin of safety and deserves special mention. This, like so many other agents, can now be safely and advantageously given by drip infusion, and has a wide application for all hypertonic states of the uterus. A suitable solution is 300 milligrams of pethidine in 1,000 milliliters of glucose saline. Individuals vary in their sensitivity so the infusion should be commenced cautiously and the speed of administration thereafter varied to suit the patient, who can be roused or put to sleep by turning the stopcock. Although rarely necessary, as much as 600 to 800 milligrams can be given within 24 hours without apparent ill effect on either mother or child. There have been recent reports of some women exhibiting an idiosyncrasy to pethidine (Zuck, 1951; Reynard, 1951), but considering its widespread use, this must be rare. During the 3 years we have been using the above technique we have not encountered untoward reactions.

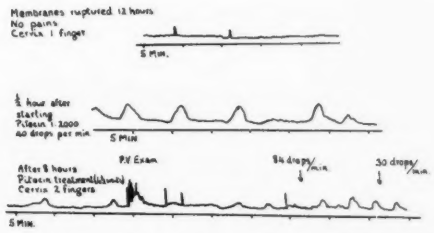

Fig. 6. Tocograph illustrating good initial response to oxytocin but the uterus becoming refractory after 8 hours' treatment. The third tracing shows very little effect even when speed of infusion is increased to 84 drops per minute. F. J., aged 31 years, primigravida; uterine inertia; cesarean section after 33 hours.

Neither morphia nor pethidine have any significant inhibitory effect on normal uterine action in labor. They cannot send a woman out of labor as some obstetricians still fear (Fig. 8). They do sometimes, however, quiet the inco-ordinate uterus and when their influence is later withdrawn uterine behavior may be more normal in character (Figs. 7, 9). This effect cannot be guaranteed, but they always give a patient a prolonged period of rest, remove any possible nervous inhibition

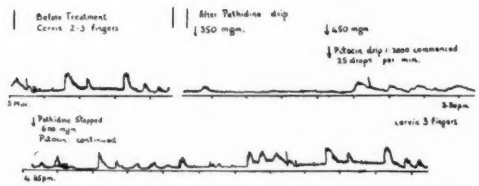

Fig. 7. Inco-ordinate uterus shown here by contractions varying in time and force. Activity almost completely suppressed after prolonged intravenous pethidine infusion. Activity restored to some extent by giving oxytocin with pethidine in the glucose saline. Ultimate effect of treatment unsatisfactory. Contractions still inco-ordinate when they returned. The action of oxytocin is to increase whatever pattern of activity is already present. E. C., primigravida; cesarean section after 72 hours' labor.

of the uterus, and give more time for a spontaneous cure to take place.

In certain cases there may be a place for a combination of pethidine and oxytocin therapy, or continuous intravenous oxytocin may be given after hypertonus has been reduced by prolonged pethidine therapy.

Other antispasmodics such as magnesium sulfate, adrenalin, amyl nitrite, nitroglycerine, and proprietary preparations are of limited or negligible value except for temporary relaxation of a constriction ring.

Sympathetic depressants. Tetraethyl ammonium chloride, given with the idea of suppressing an overactive sympathetic nervous system, is of no real practical value in inco-ordinate uterine action. It appears to affect the uterus by way of causing more prolonged and frequent contractions for 1 hour following its intravenous injection. But it does not change the pattern of activity or facilitate dilatation of the cervix (Jeffcoate, 1949). Dihydroergotamine has also been used for its

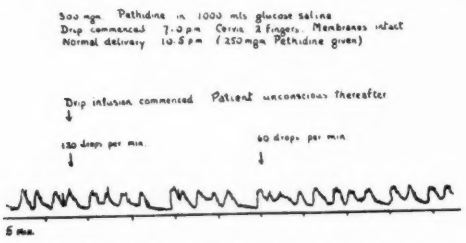
300 mgm Pethidine in 1000 mls glucose saline
Drip commenced 7.0 p.m Cervix 2 fingers. Membranes intact
Normal delivery 10.5 p.m (250 mgm Pethidine given)
Drip infusion commenced
Patient unconscious thereafter
120 drops per min.
60 drops per min
5 min.

Fig. 8. Record of normal labor showing that intravenous pethidine, even when given in doses large enough to keep patient unconscious, does not allay normal uterine action. T. J., primigravida.

sympathicolytic effect with reported good effect (Gill and Farrar, 1951). We employed it by continuous intravenous drip infusion using concentrations varying from 1 to 3 milligrams per 1,000 milliliters. It has no effect on the uterus in late pregnancy or in normal labor (Fig. 10), but in inco-ordinate uterine action it appears to increase the frequency, but not amplitude, of contractions, and it does not lower tone as is claimed (Figs. 11, 12). We have never seen it benefit the progress of labor.

Nerve block. Interruption of the autonomic nerve pathways by paravertebral block of the sympathetic ganglia, paracervical injection of Frankenhauser's plexus and caudal anesthesia is also reported to give good results, but we have no experience of it. It is now claimed (Arthur and Johnson, 1951) that continuous low caudal anesthesia, so low that it interrupts parasympathetic rather than sympathetic nerve connections, improves uterine polarity, and favors dilatation of the cervix in these cases. If these findings and their explanation are confirmed, the present concept of the innervation of the uterus may require revision.

Estrogens. Estrogen therapy, which was at one time advocated for the treatment of hypotonic inertia (Jeffcoate, 1938), has not fulfilled early expectations. And this is not surprising, since the theoretical basis of the treatment is insecure. Further experience leads to the conclusion that estradiol, even when given intravenously in massive doses, does not significantly influence the normal, the inert, or the inco-ordinate uterus in labor.

Operative treatment. Once the cervix becomes fully dilated or if uterine action only becomes faulty in the second stage of labor, and disproportion being excluded, forceps delivery is indicated without delay. It is wrong to tax the resources of the woman to the limit, or to wait for evidence of fetal asphyxia. The operation is often difficult, however, because the presenting part has to be delivered from the midcavity and has first to be rotated from transverse or occipitoposterior position.

The real clinical problem, however, arises when some circumstance calls for delivery of the child before the cervix is fully dilated.

Cervicotomy followed by forceps delivery has a small but definite place in such circumstances. It should, however, only be undertaken when the fetal head is presenting and is fully engaged, and when the cervix is completely effaced, thin, and at least half dilated. It is indicated therefore in cervical dystocia and not in inco-ordinate uterine action. The literature contains reports of varied experiences with incision of the cervix; some authorities condemn the operation outright, others write enthusiastically of it. The results, however, depend on a proper selection of cases. If cervicotomy is carried out when the upper cervix is not effaced or is in spasm, when the fetal head is high and when the uterus as a whole is inefficient, it is full of danger for both mother and child. If it is restricted to the treatment of true cervical dystocia, it is an easy, safe, and satisfying operation.

There has also been some dispute as to whether the incisions should be sutured when delivery is complete, and surprise has been registered that regardless of suturing, the cervix later shows no more sign of injury than does the usual multiparous organ. The explanation is that if the case is well chosen the incisions are still confined to what is, in the nonpregnant cervix, the neighborhood of the external os, and when involution of the thin and stretched cervix is complete, a 1 to 1½ inch incision becomes no more than a nick in the external os.

In all cases except cervical dystocia, however, which means in the majority, incomplete dilatation of the cervix is best circumvented by lower segment cesarean section, an operation which for the present must hold an important place in the treatment of severe degrees of faulty uterine action arising in the first stage of labor. Without it fetal and maternal morbidity rates are inevitably high.

No matter what form of vaginal or abdominal operative treatment is undertaken, however, one of the greatest risks in these cases arises from anesthesia. The stomach is distended, and food taken 12 hours earlier remains undigested. Fluid is in the stomach even if nothing has been taken by mouth. With anesthesia vomiting is extremely likely,

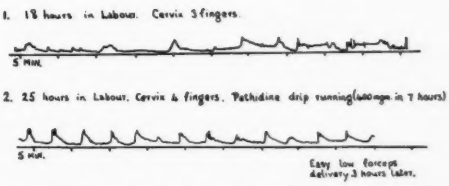
1. 18 hours in Labour. Cervix 3 fingers.
5 min.
2. 25 hours in Labour. Cervix 4 fingers. Pethidine drip running (100mgs in 7 hours)
5 min.
Easy low forceps delivery 3 hours later.

Fig. 9. Tocograph of case of minor degree of inco-ordinate uterine action showing an apparent beneficial effect of intravenous pethidine. Second tracing taken 7 hours after commencing treatment and showing that contractions are now more regular and forceful. D. B., primigravida, aged 31 years, inco-ordinate uterus. Membranes ruptured at onset of labor; good progress to 3 fingers dilatation in 6 hours when irregular back pains supervened.

and inhalation of stomach contents is a significant cause of maternal death. Although local analgesia is theoretically the ideal, few of these nervously exhausted women can tolerate it and some form of general anesthesia, with adequate safeguards, is generally necessary.

RESULTS OF TREATMENT

Immediate. The dangers of inefficient uterine action are those of prolonged labor, and the maternal and fetal morbidity rates rise in an ever-steepening curve once labor has lasted more than 24 hours. It is not easy to compare statistics because they vary considerably according to the types of cases under review. For example, the outlook in mild or moderate degrees of inertia is very different from that in constriction ring dystocia.

During the first quarter of this century the literature suggests that in serious inco-ordinate uterine action, as in constriction ring dystocia, the maternal death rate varied between 10 and 50 per cent, and the fetal mortality was between 40 and 80 per cent. The situation has changed materially in the last 20 years, and most reports indicate that for all types of uterine disorder of severe degree the maternal and fetal mortality rates are not more than 1 and 15 per cent respectively.

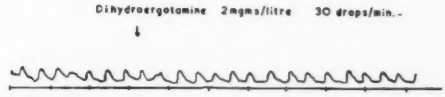

Fig. 10. Dihydroergotamine intravenous infusion during normal labor. No effect on uterine action. First stage of normal labor in primigravida No. 114509.

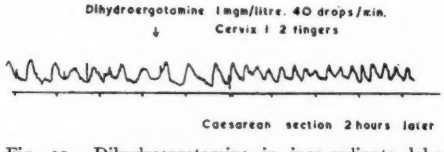

Fig. 11. Dihydroergotamine in inco-ordinate labor. Contractions increased in frequency but not amplitude, and a suggestion of increased tone in uterine wall. Mrs. C., No. 13637, primigravida, aged 27 years. Cesarean section after 30 hours in labor.

Moreover, the results continue to improve as is illustrated by our two series of cases, one reported in 1949 and one collected since then (see Tables VII and VIII). The total of 277 cases (279 babies) referred to previously is made up of these two groups.

The improvement in the fetal salvage in more recent years can be attributed mainly to the increasing attention paid to faulty uterine action, which resulted in every case being carefully watched and assessed. Modifications in treatment included more timely and skillful forceps deliveries, and an increased use of cesarean section. Except for cesarean section the fetal mortality in either of the above series would not have been less than 50 per cent.

The fact that there were no maternal deaths in either of these series does not mean that the condition has ceased to have its dangers for the mother. Many of the women, especially in the first series, were seriously ill, and suffered extensive soft tissue laceration, shock, and puerperal infection.

Remote. It is of some importance to know the ultimate effect on the childbearing function of women who pass through the ordeal of painful and exhausting labor, for it may influence the outlook on treatment. We have therefore followed up hospital cases who suffered inco-ordinate uterine action 3 to 13 years previously. All those delivered less than 3 years ago are excluded and only the most severe cases—those which required cesarean section or midforceps delivery—are accepted. Many of these old patients can no longer be traced but we have details of 147 consecutive followed-up cases and the findings are shown in Table IX.

The striking feature of these figures is that only about half the patients have so far conceived again, and in more than one third of the cases the infertility is voluntary. Although domestic circumstances sometimes played a part in the decision to practice contraception, in the majority of cases it was the experience of the first labor which prompted the wife or husband to avoid further pregnancies. It was hoped that this enquiry might give some indication as to whether a labor of moderate length ending in cesarean section is more disconcerting than a rather longer

TABLE VII

	No. of cases	No. of babies born	Maternal deaths	Still-births	Neo-natal deaths	Fetal mortality per cent
Private and hospital cases reported 1949	101	102	0	9	4*	12
Hospital cases (L.M.H. and M.R.H. 1949, 1950, 1951)	176	177	0	4	1	3

*1 child died from fetal malformation.

TABLE VIII

Mode of delivery	Hospital and private practice 1946 to 1948 series			Hospital only 1949 to 1951 series		
	No. of babies born	Still-births	Neo-natal deaths	No. of babies born	Still-births	Neo-natal deaths
Spontaneous delivery	22	1		15		
Forceps with or without rotation	38	3	3	58	2°	1
Cervicotomy and forceps	5	2†		2†	1	
Failed forceps spontaneous delivery	1					
Failed forceps internal version	1	1				
Lower segment cesarean section	35	2	1*	102‡	1	
Total	102	9	4	177	4	1

*1 died from fetal malformation.
†1 in each group died in utero from asphyxia before operation.
‡1 set of twins in each group.

TABLE IX

	No. cases	Per cent
Cases (80) treated by cesarean section		
Further conception avoided	33	41
Involuntary sterility (2 years or more)	8	
Further pregnancies	38	
Died from intercurrent disease	1	
Cases (67) treated by forceps delivery		
Further conception avoided	25	37
Involuntary sterility (2 years or more)	5	
Further pregnancies	37	

TABLE X*

Mode of delivery in second pregnancy	Cesarean section in first pregnancy No. patients	Forceps delivery in first pregnancy No. patients
Still undelivered	1	2
Abortion	2	1
Spontaneous delivery	6	26
Forceps delivery	3	4
Elective cesarean section	19	2
Trial labor, cesarean section	7†	2
Total	38	37
Total of subsequent pregnancies among all patients at time of follow-up	43	45

*Details of cases shown in Table IX.
†One of these had a spontaneous delivery in the 3rd pregnancy.

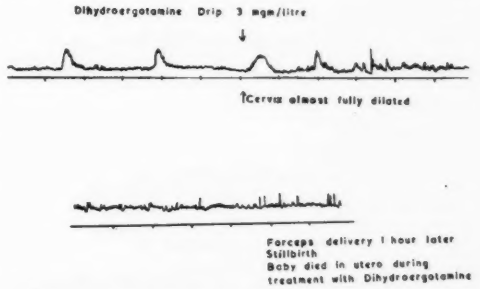

Fig. 12. Inco-ordinate uterine action. Again the effect of dihydroergotamine is to raise tone and to cause frequent small contractions. The death of the fetus did not appear to be related to the giving of dihydroergotamine but rather to an error in judgment, the application of forceps being unnecessarily delayed. Mrs. B., primigravida, aged 26 years; 48 hours in labor before treatment.

labor ending in forceps delivery, and as to which treatment is better from the standpoint of the ultimate size of the family. There is little difference in the figures, and further analysis suggests that the future outlook of the patient is influenced more by the fate of the child than the type of treatment. Maternal instinct proved stronger than the fear of another labor and all except 1 of those women who avoided conception had a living child. A few only embarked on pregnancy again on being promised that subsequent delivery would be by elective cesarean section.

When women are prepared to face pregnancy again, how does the uterus behave in subsequent labors? Tables X and XI suggest the answer to this question. Table X gives details of cases analyzed in Table IX, while Table XI includes the same cases together with others collected from private practice. Providing it is given the chance, the uterus is usually more efficient in the next labor. A difficult forceps delivery at the end of a prolonged labor is nearly always followed by a rapid and spontaneous delivery next time. When cesarean section is necessary in the first labor, the outcome of the second is largely, but not entirely, governed by the degree of dilatation of the cervix at the time of the operation. If it is less than half dilated another section may be necessary despite the fact that the cervix attains further dilatation. If, however, the cervix is half or more dilated the first time, there is a good chance it will reach full dilatation in the next labor. Seventeen out of 28 women allowed to go into a second labor, having been delivered by cesarean section previously, had a vaginal delivery. Women who have cesarean section in the first labor and forceps delivery in the second, usually achieve spontaneous delivery in the third. This might be described as the typical history.

The natural tendency for the uterus to function better with each delivery is an important consideration when deciding treatment. To carry out cesarean section the first time does not necessarily condemn the woman to several repeat sections. On the other hand, it is usually unnecessary to carry out an elective cesarean section for a "bad obstetric history" which consists of no more than a single prolonged labor ending in forceps delivery and stillbirth. This probably applies to 3 cases shown in Table XI. It might be argued that the natural history of uterine dysfunction warrants taking a risk with the first child, at

TABLE XI

Mode of delivery in second pregnancy	Cesarean section in first pregnancy No. patients	Forceps delivery in first pregnancy No. patients
Spontaneous delivery	8	38
Forceps delivery	9	5
Elective cesarean section	24	3
Trial of labor, cesarean section	11*	—

*In 3 cases labor lasted less than 12 hours, cesarean section then being carried out for indications other than faulty uterine action. Another patient had a normal delivery in her third pregnancy.

any rate in a young woman. It may be said that it is worth even sacrificing the first child to achieve a vaginal delivery—in the knowledge that the patient will have future children without difficulty. There are, however, fallacies to this argument. The risk to the first child may be permanent injury rather than death, the mother herself may be seriously injured and, as the figures show, she may not have any more pregnancies.

THE CAUSES OF INEFFICIENT UTERINE ACTION

Brief consideration of the causes of inefficient uterine action serves in many respects to summarize what has gone before. The comments are based on a study of severe uterine disorders in which the diagnosis was not in doubt, but they are applicable to the more common minor dysfunctions. It can be stated with a fair degree of confidence that:

1. The condition is rarely, if ever, due to overstretching of the uterine wall as in twins and hydramnios. Nor is it a characteristic of the worn out and fibrous uterus of the grande multipara. In fact this type of uterus is so efficient that it ruptures itself spontaneously on the slightest provocation. Diseases of the uterine wall such as fibroids are not a significant cause. Faulty development of the uterus, such as bicornuate uterus, can operate, but this type of deformity is present in only a small proportion of cases.

2. The condition is essentially a disease of primigravidas, at least 95 per cent of severe cases occurring in first labors. Moreover, it is not the prerogative of elderly primigravidas, the age incidence being only slightly higher than the age of first childbearing in any community. It is perhaps rather more common in the heavily built, thickset, and relatively infertile woman, and can form part of the dystrophia dystocia syndrome. Obesity alone seems to favor it. It also sometimes appears to have a familial incidence, but it is doubtful whether this implies an inherent defect, or whether it is the result of fear or other emotion induced by the experience of a close relative.

3. The condition is often caused by or associated with a minor degree of disproportion and, in particular, with a posterior position of the occiput or a large fetus. The association between a high presenting part and faulty uterine action, in the absence of disproportion, raises the question as to whether the failure of engagement is the cause or the result of the particular behavior of the uterus. In other words the head may remain high because hypertonicity of the lower segment is already present before labor. False labor, too, can be regarded as representing inefficient uterine action.

4. Primigravidas, it is generally said, are prone to inco-ordinate uterine action and inertia because they are fearful of an unknown experience. Fear, acting by the liberation of adrenalin or by the sympathetic nervous system, leads to tension in the lower segment and upper cervix. Elimination of fear by antenatal education in the physiology of childbirth and in mental and physical relaxation ensures, it is said, smooth, rapid and less painful labor. There is a large body of opinion and some evidence in support of this view, and there can be little question that emotional upsets can cause minor disturbances and variations in the behavior of the uterus in labor. When we come to the more severe and serious inertia or inco-ordinate activity of the uterus, does this theory of causation remain acceptable?

The woman who suffers from these conditions is not always the fearful one—on the contrary, she is often placid, sensible, and well-informed. The obvious neurotic who might be expected to have trouble usually has none. There is no significant association between inco-ordinate uterine action and spasmodic dysmenorrhea. It has not yet been shown that drugs which depress the sympathetic nervous system can favorably influence the progress of labor when the uterus is inefficient, although there are some claims for continuous caudal anesthesia. Moreover, no one has yet shown that the emotional and physical preparation of the patient antenatally reduces the incidence of inco-ordinate uterine action, or even the duration of normal labor and the need for forceps delivery. Indeed the only evidence suggests that it makes no difference.

Leaving aside these arguments, however, there is one fundamental feature of inefficient

uterine action which throws the greatest doubt on the nervous theory of origin. No matter how afraid she may be when she faces her first confinement, the woman who experiences inco-ordinate labor finds it far worse than her worst fears led her to expect. So much is this so that at least one third of these patients refuse to face pregnancy again. Those that do go into labor a second time are full of apprehension which nothing can allay. Yet despite this the uterus nearly always behaves better, even the pain sensation is different, a point on which most patients are quick to comment. This evidence is so striking, and can be confirmed daily in practice, that it cannot be set aside.

Finally, a woman with a uterus didelphys may experience normal first labor when one horn is impregnated, only to suffer severe inco-ordinate action if the second pregnancy happens in the other horn, her emotional outlook remaining the same, or being even better after her first satisfactory experience.

The change in uterine function from one pregnancy to another seems best explained by supposing that passive rigidity, rather than temporary muscle spasm, of the tissues of the lower segment and cervix may play a greater part in inco-ordinate uterine action than has previously been recognized. If this is so then the stretching of the first labor permanently overcomes this resistance, thus allowing more normal behavior in the future.

This paper began by pointing out that the primigravid uterus is always relatively inefficient because of inherent resistance in its lower part. It may be concluded by suggesting that inco-ordinate uterine action is perhaps merely an exaggeration of what is in most women a normal or physiological degree of inefficiency.

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ASCORBIC ACID IN SURGICAL PATIENTS WITH PARTICULAR REFERENCE TO THEIR BLOOD BUFFY LAYER

I. Survey of Blood and Tissues

JOHN H. CRANDON, M.D., F.A.C.S., STANLEY MIKAL, M.D., and
BERNARD S. LANDAU, Ph.D., Boston, Massachusetts

DESPITE the knowledge that complete deficiency of vitamin C in man results in failure of wound healing, the physiological significance of varying amounts of ascorbic acid in the different components of the blood and tissues has not been entirely clarified.

When interest in the ascorbic acid content of human blood first developed, plasma levels below 0.7 milligram per cent were considered to indicate a subclinical scorbutic state. With more careful observation, however, it became evident that much lower plasma and whole blood levels were compatible with adequate vitamin C nutrition. In 1940 Butler and Cushman (2) concluded that the buffy layer or white cell-platelet layer content of ascorbic acid, rather than the plasma or whole blood content, was the best index of the body status with respect to the vitamin in the absence of infection and leucemia. This has been confirmed by Pijoan and Lozner and Lowry and associates. These conclusions, however, were not based on tissue analyses. Attempt to correlate the buffy layer and tissue ascorbic acid with the condition and response of patients suffering from various surgical conditions would thus seem warranted, particularly since more recent experimental investigations have indicated a marked fluctuation of adrenal ascorbic acid and variation of response to administration of ascorbic acid in animals during various phases of the alarm reaction.

To this end, diverse types of surgical cases exclusive of burns were studied during the past 2 years at the Boston City Hospital. The great majority of these patients were seriously ill. Slightly over 600 white cell-platelet or buffy layer determinations and over 800 determinations of both plasma and whole blood samples, as well as many urine analyses, were performed. Blood samples were drawn for the most part in the morning without the patient necessarily in a fasting state, since the hospital breakfast is completely devoid of vitamin C. Analysis of the regular unsupplemented daily diet of the hospital showed a total content of no more than 10 milligrams of vitamin C, so that the intake by consumption of food was an almost negligible factor in these studies. Tissue determinations were performed on 70 patients. Aside from various special tissues studied, samples were taken generally from the anterior rectus sheath and underlying muscle in amounts of not less than ½ gram. Immediately after collection all samples were analyzed by the Roe and Kuether method and the white cell-platelet layer was isolated by the method of Butler and Cushman (3).

CRITIQUE OF METHOD

Analysis of the buffy layer ascorbic acid was both difficult and tedious, each determination requiring approximately 6 hours. Following centrifugation of the blood sample in a Cushman tube with specially constricted stem, the supernatant buffy layer was found actually to consist of 2 layers—an uppermost ivory-white layer and a lower, slightly red-tinged layer. Contamination of the uppermost layer with any of the lower red-tinged layer led to a higher reading, and was avoided. The difficulty in withdrawing only this supernatant ivory-colored layer of the buffy coat has led others to profoundly distrust the figures on buffy coat analyses (11).

Analysis of the plasma, whole blood, and tissue ascorbic acid, on the other hand, was

From the First Surgical Service and the Surgical Research Laboratory, Boston City Hospital, and the Department of Surgery, Tufts College Medical School. This study was made possible by 2 grants from the Hoffmann-LaRoche Company of Nutley, New Jersey.

found to be relatively simple and devoid of pitfalls leading to error.

RESULTS

The average ascorbic acid levels in the buffy layer, whole blood, and plasma of 20 normal persons together with the range of values and the calculated standard deviation are presented in Table I. Ten individuals were tested in October and 10 in February. Considerable variation, particularly in the buffy layer levels, is evident. The values in the winter month group are somewhat lower than those determined in the fall months.

The results of blood determinations on 200 consecutive surgical patients who received no known vitamin C beyond that contained in the regular hospital diet are tabulated in Table II. They have been arbitrarily arranged in groups of 20 to expose "drifting" and seasonal variations. From the range of values, a considerable variation in the buffy layer ascorbic acid level is again evident. As with normal individuals, the average levels tend to fall somewhat during the winter months with a return to the highest values in May. This seasonal fluctuation has been found previously in the plasma of fasting individuals by others (8).

The over-all averages for the blood levels of the 20 normal individuals were 15.2 milligrams per 100 grams of white cell-platelet layer, 0.73 milligram per cent of whole blood, and 0.69 milligram per cent of plasma. For the 200 consecutive surgical patients, these average values were 14.5 milligrams per 100 grams, 0.48 milligram per cent, and 0.36 milligram per cent, respectively. Thus, whereas the average white cell-platelet ascorbic acid levels for normal individuals and surgical patients are essentially the same, the average whole blood and plasma levels of the Boston City Hospital surgical patients are considerably lower than those of normal persons. Bartlett, Jones, and Ryan found an average of 0.43 milligram per cent in the plasma of 188 surgical patients at the Massachusetts General Hospital.

Comparison of the plasma and whole blood ascorbic acid values of individual surgical patients reveals that there is an almost linear relationship between the two, the whole blood being almost constantly slightly higher than that of the plasma in this series. A similar finding was reported by Roe and Kuether studying 50 surgical patients (10).

When the plasma and whole blood ascorbic acid content is compared with that of the white cell-platelet layer, no such correlation is found. Nevertheless, a certain interrelationship is seen to exist. In over 600 surgical patients studied, the only ones showing a high plasma ascorbic acid (above 0.4 milligram per cent) with a concomitantly low buffy coat ascorbic acid (below 7 milligrams per 100 grams) were the few deficient patients who had been given large doses of ascorbic acid intravenously shortly before their blood was analyzed. In all others, if the plasma content was high, the buffy layer content of ascorbic acid was also high. On the other hand, low plasma and whole blood ascorbic acid values were frequently associated with moderate buffy layer levels.

In 70 cases, in addition to the blood determinations, sizable biopsies (0.5 gram or more) were taken from the operative wounds and, from this tissue, muscle and fascia were carefully separated and determinations performed on one or both. Results are listed in Table III.

Upon analysis of these data, it was found that the majority of patients who had rectus fascia which showed an ascorbic acid content below 1.5 milligrams per 100 grams also had low blood levels; and that all but 3 patients in this group had had a low intake of ascorbic acid relative to the severity of their disease state. On the other hand, among those patients whose fascial values of ascorbic acid ranged above 1.5 milligrams per 100 grams, no consistent proportionate dietary intake or blood content of the vitamin could be demonstrated.

Even in the fascia low level group, no direct relationship between tissue and blood levels in individual patients could be found. The plasma rather than the buffy layer showed more consistently low values, indicating that the plasma reflects more accurately the tissue status than does the buffy layer when the tissues are deficient in ascorbic acid. Only 3 patients of this low level group had plasma

TABLE I.—BLOOD ASCORBIC ACID LEVELS OF 20 NORMAL INDIVIDUALS ON AN AVERAGE DIET

Period of 10 determinations	White cell-platelet layer mgm./100 gm.			Whole blood mgm. per cent			Plasma mgm. per cent		
	Range	Average	Standard deviation	Range	Average	Standard deviation	Range	Average	Standard deviation
Oct.–Nov.	10–50	19.3	11.80	0.50–1.05	0.83	0.16	0.61–1.06	0.82	0.14
Feb.–Mar.	4.1–25	12.2	6.35	0.24–1.03	0.62	0.28	0.10–1.00	0.55	0.32
Over all	4.1–50	15.2	9.07	0.24–1.05	0.73	0.22	0.10–1.06	0.69	0.23

levels over 0.18 milligram per cent, and each of these had been receiving a regular daily ascorbic acid supplement for severe disease as follows: E.B., acute cholecystitis with pancreatitis, 100 milligrams daily; H.C., severe recurrent ulcerative colitis, 300 milligrams daily; C.R. carcinomatosis, peritonitis, and wound dehiscence, 1,000 milligrams daily.

Certain other cases in this group merit brief comment. Two patients, J.K. and O.J., showed a clinical picture of scurvy, with multiple ecchymoses over the skin. The former patient, however, was in the hospital only 24 hours before testing, so that the appreciable buffy layer content may have been the result of ascorbic acid intake not detected in his dietary history. The other patient, a 72 year old female having extensive pulmonary tuberculosis, tuberculosis of the cecum, and pernicious anemia, received only 50 milligrams of ascorbic acid daily while she was on the medical ward for 4 weeks prior to surgery. She showed a typical picture of scurvy and very low values at the first operation, and zero readings in both buffy layer and tissues at time of prophylactic femoral vein ligation 4 days later. She was immediately placed on 200 milligrams of ascorbic acid daily, and subsequently her wounds healed satisfactorily. E.P., a 52 year old epileptic taking dilantin regularly, had been on a strict diet devoid of fruit in any form for years. Although the fascial ascorbic acid content was zero and the plasma very low, the buffy layer ascorbic acid was only slightly subnormal. Following a herniorrhaphy, this patient's wound showed good healing in spite of no postoperative supplement of ascorbic acid, and there has been no recurrence of his hernia after a period of 1 year. Similarly, all other patients in this group having an extremely low value of

TABLE II.—BLOOD ASCORBIC ACID LEVELS OF 200 CONSECUTIVE GENERAL SURGICAL PATIENTS RECEIVING NO KNOWN VITAMIN SUPPLEMENT

Period of 20 determinations	White cell-platelet layer mgm./100 gm.			Whole blood mgm. per cent			Plasma mgm. per cent		
	Range	Average	S. D.	Range	Average	S. D.	Range	Average	S. D.
Oct. 2–Nov. 1	7.0–37.5	15.5	7.08	0.15–0.80	0.42	0.21	0.11–0.65	0.35	0.15
Nov. 2–Nov. 17	3.2–32.2	12.0	7.95	0.03–1.05	0.40	0.26	0.03–0.9	0.30	0.25
Nov. 18–Dec. 13	2.9–32.0	16.2	9.40	0.12–0.84	0.47	0.22	0.08–0.71	0.33	0.21
Dec. 14–Jan. 10	0.0–30.0	11.2	7.92	0.0 –0.97	0.30	0.26	0.03–1.13	0.28	0.26
Jan. 11–Feb. 15	0.0–34.3	9.1	8.31	0.06–1.33	0.48	0.36	0.03–1.19	0.37	0.31
Feb. 16–Mar. 28	0.0–20.8	9.6	5.33	0.12–1.50	0.54	0.34	0.06–1.53	0.30	0.33
Mar. 29–May 2	3.1–30.9	16.6	7.31	0.06–1.33	0.44	0.27	0.0 –1.27	0.37	0.21
May 3–June 4	7.5–53.7	25.6	13.78	0.14–2.03	0.75	0.47	0.13–1.44	0.58	0.36
June 5–July 12	0.0–49.0	17.6	11.10	0.17–1.35	0.74	0.35	0.09–1.23	0.51	0.32
July 13–July 27	1.6–27.0	11.6	5.97	0.13–1.30	0.36	0.25	0.05–0.99	0.23	0.22
Over all	0.0–53.7	14.5	8.4	0.0 –2.03	0.48	0.30	0 0 –1.53	0.36	0.26

S.D.—Standard deviations.

TABLE III.—CONCENTRATION OF ASCORBIC ACID IN TISSUE AND BLOOD OF 70 SURGICAL PATIENTS (ROE AND KUETHER METHOD)

High level in fascia 6–15 mgm./100 gm.						Medium level in fascia 5–1.5 mgm./100 gm.						Low level in fascia 1.4–0 mgm/100 gm.					
Name	Fascia	Muscle	Buffy layer	Whole blood	Plasma	Name	Fascia	Muscle	Buffy layer	Whole blood	Plasma	Name	Fascia	Muscle	Buffy layer	Whole blood	Plasma
G.G.	10	5	20.8	0.36	0.26	W.F.	3.0		5.6	0.17	0.09	D.C.	0.4	0.5	4.0	0.17	0.09
A.O.	6		16.6	0.28	0.24	C.L.	5.0		9.6	0.47	0.34	C.W.	0.7	0.7	6.5	0.27	0.10
M.M.	11		12.5	0.46	0.30	D.F.	1.9	0.4	23.0	0.47	0.32	J.K.	0.5	1.0	7.5	0.21	0.09
W.D.	8	2	19.7	0.33	0.45	M.R.	5.0	8.0	53.7	0.58	0.40	E.P.	0.0	5.0	13.2	0.35	0.16
G.C.	8		23.9	0.64	0.59	F.I.	4.0	2.0	50.0	1.38	1.27	F.R.	1.3		3.1	0.13	0.0
C.D.	10	6	11.5	0.44	0.38	G.G.	4.1		29.3	0.86	0.84	L.G.	1.4		11.7	0.19	0.07
G.T.	10		33.7	0.43	0.27	J.G.	3.0	1.0	7.5	0.14	0.13	L.W.	0.5	0.0	5.6	0.23	0.08
S.G.	8		10.4	0.26	0.16	M.R.	5.0	2.0	31.2	1.05	1.2	J.C.	1.0	2.0	3.7	0.21	0.12
J.C.	9	3	9.8	0.27	0.23	M.J.	5.0	3.0	18.2	1.13	0.76	S.K.	0.9	1.0	2.5	0.09	0.0
M.H.	9	3	23.6	0.50	0.40	J.B.	3.0	2.0	57.2	1.28	1.38	O.J.	0.4	1.0	3.4	0.14	0.09
E.R.	6	2	49.0	0.59	0.55	W.O.	3.0	4.0	22.1	1.16	1.21	O.J.	0.0	0.0	0.0	0.12	0.09
J.R.	10	3	68.7	1.41	1.44	J.K.	3.0	2.0	19.5	0.86	0.60	C.R.	0.0	0.7	1.6	0.01	0.06
C.C	15	4	8.3	0.23	0.12	W.B.	3.0	6.0	20.8	0.46	0.56	E.L.	0.0	0.0	0.0	0.27	0.07
P.C.	10	5	23.3	0.95	0.87	T.R.	3.0	2.0	20.5	0.41	0.34	J.R.	1.4	0.6	1.1	0.13	0.11
J.M.	8	6	11.5	0.77	0.70	T.G.	4.0	1.0	27.0	0.56	0.45	L.E.	0.8	0.5	11.7	0.22	0.18
M.G.	12	3	24.3	0.75	0.70	J.M.	3.0	2.0	11.2	0.47	0.30	J.W.	0.1	0.1	2.8	0.19	0.07
H.B.	9		19.3	0.78	0.80	M.P.	2.0	2.0	16.1	0.56	0.54	L.E.	1.2	0.9	0.0	QNS	0.0
						S.W.	3.0	9.0	18.7	1.3	0.99	E.B.	0.7		2.2	0.14	0.02
						J.W.	3.0	0.8	22.5	0.27	0.06	C.D.	0.0	0.3	5.0	0.31	0.12
						F.Y.	3.0	1.0	14.1	0.13	0.12	H.C.	1.0	0.8	9.5	0.43	0.31
						B.D.	3.0		6.5	0.40	0.37	E.S.	0.7	1.0	9.3	0.12	0.11
						N.V.	4.0		7.3	0.38	0.29	S.M.	0.0	0.2	2.9	0.10	0.04
						S.M.	2.0	0.9	7.0	0.36	0.33	E.B.	0.5	1.0	10.7	0.35	0.36
						A.C.	2.0	1.0	24.3	0.51	0.31	J.F.	0.9	1.0	8.8	0.21	0.15
						S.H.	3.0	3.0	19.8	2.9	3.0	J.D.	0.0	0.2	8.3	0.19	0.12
						J.M.	1.7	2.8	20.6	0.60	0.40	C.R.	1.4		9.9	0.47	0.43
						P.F.	5.0	3.0	19.0	0.44	0.25						
						R.S.	2.0	3.0	13.0	QNS	1.03						

Fascia and muscle: mgm./100 gm; buffy layer: mgm./100 gm.; whole blood and plasma; mgm. per cent

ascorbic acid in both fascia and plasma but appreciable quantities in the buffy layer showed clinically adequate wound healing. Further inquiry into the relationship between buffy layer ascorbic acid content and wound healing will be reported subsequently.

One patient, L.E., a 62 year old chronic alcoholic undergoing a two stage lumbar sympathectomy for frostbite of the toes, showed a slight rise in ascorbic acid content of the fascia at second operation, but a fall of the buffy layer level from 11.7 milligrams per 100 grams at first operation to zero at the second. He received no ascorbic acid supplement at any time. Although the first operative wound healed well, 7 days after a second sympathectomy a small portion of the second wound, when subjected to tension, pulled apart easily. His convalescence was otherwise uneventful.

Of the remaining patients in this group showing extremely low ascorbic acid in both fascia and buffy layer, all but 2 were placed on supplements of the vitamin ranging from

TABLE IV.—ASCORBIC ACID CONTENT OF BLOOD AND VARIOUS TISSUES IN SURGICAL PATIENTS

Patient	Disease	Buffy layer	Whole blood	Plasma	Tissues
		Mgm./100 gm.	Mgm. per cent		Mgm./100 gm.
J.K.	Cholecystitis	19.5	0.86	0.60	Fascia 3.0; Muscle 2.0; Appendix 17.0; Gallbladder 8.0
M.G.	Ruptured follicle	24.3	0.75	0.70	Fascia 12.0; Muscle 3.0; Appendix 16.0
F.O'C.	Duodenal ulcer	22.1	1.16	1.12	Fascia 3.0; Muscle 4.0; Stomach wall 5.0
H.J.	Gastric ulcer	28.6	0.68	0.51	Fascia 2.0; Muscle 4.0; Ulcer 14.0; Stomach wall 12.0
J.D.	Gastrectomy	8.3	0.19	0.12	Fascia 0.0; Muscle 0.2; Stomach (cardia) 3.0; Duodenum (scar) 2.0; Jejunum 5.0
E.B.	Varicose veins	2.2	0.14	0.02	Fascia 0.7; Vein 0.0
C D.	Varicose veins	11.5	0.44	0.38	Fascia 10.0; Muscle 6.0; Vein, Artery 5.0
C.W.	Benign hypertrophy of prostate following ruptured appendix and wound sepsis	6.5	0.27	0.10	Fascia 0.7; Muscle 0.7; Prostate 3.0; Bladder 1.0
C.DeF.	Herniorrhaphy and orchidectomy	5.0	0.31	0.02	Fascia 0.0; Muscle 0.3; Testicle 10.9
H.H.	Fibrosarcoma, thigh	13.2	0.31	0.29	Fascia 0.7; Muscle 0.9; Cancer 6.0
S.McC.	Carcinoma of sigmoid	7.0	0.36	0.33	Fascia 2.0; Muscle 0.9; Colon 3.0; Cancer 10.0
J.M.	Evisceration	11.5	0.77	0.70	Granulations; Fascia 8.0; Muscle 6.0
E.McL.	Draining sinus following pneumonectomy with insertion of plastic sponge	14.3	0.54	0.35	Fascia 1.3; Muscle 1.8; Granulation tissue 8.4

50 to 200 milligrams a day postoperatively and enjoyed uneventful convalescence. Two patients, C.W., and J.W., with low ascorbic acid levels in both fascia and buffy layer at the time of surgery, did very poorly during operation and died 72 and 24 hours, respectively, thereafter. The only positive finding in one at autopsy was interstitial myocarditis with focal necrosis. The only findings in the other patient at autopsy were cirrhosis of the liver and generalized oozing into the wound. The low blood and tissue levels in this latter patient at operation indicated extreme deficiency since he had been given 1,300 milligrams of ascorbic acid during the 24 hours immediately preceding, in an effort to correct a postulated scorbutic state. The clotting time of this patient was not impaired. Twenty-four hours after operation and just before death, the blood ascorbic acid was essentially unchanged: buffy layer, 2.0 milligrams per 100 grams; whole blood, 0.15 milligram per cent; and plasma, 0.008 milligram per cent. Although careful review of the pathological sections failed to reveal any changes pathognomonic of scurvy, no adequate cause of death could be found in either of these cases. The possibility thus remains that ascorbic acid deficiency may have been an important factor in this outcome. Interstitial myocarditis with focal necrosis, as reported in the first of these cases, has been described as a cause of sudden death in scorbutic guinea pigs (6).

One patient, C.R., suffering from carcinomatosis, peritonitis, and wound dehiscence, at second operation showed remarkably low ascorbic acid content in blood and tissues in view of the fact that he had received over 8,000 milligrams of the vitamin parenterally during the preceding 10 days. Unfortunately, excretory studies were not performed over this period, so that it is not clear whether the findings in this case represent increased excretion, utilization, or destruction of the vitamin. This phenomenon of disappearance of large doses of ascorbic acid in certain extremely ill patients has been previously reported by Lund and associates.

Among patients with fascia ascorbic acid levels ranging above 1.5 milligrams per 100

grams, no consistent proportionate level of dietary intake or of blood content of this vitamin could be demonstrated. Attempts to correlate fascia and muscle content with either buffy layer or plasma content in the individual patient also failed in this group. However, only 2 patients in the fascia high level group showed a plasma ascorbic acid content below 0.2 milligram per cent. Both these patients, S.G. and C.C., had bleeding duodenal ulcers and were on the ward without ascorbic acid supplement for 2 weeks and 5 days, respectively, prior to gastrectomy. In the fascia medium level group, only 4 patients showed a plasma ascorbic acid content below 0.2 milligram per cent. Again, 1 of these, J.G., with a bleeding duodenal ulcer, was on the medical ward for 23 days without ascorbic acid supplement prior to operation and sampling of tissues. The other 3 patients—W.F., having a ventral hernia, J.W., a postphlebitic ulcer, and F.Y., a carcinoma of the rectum with widespread metastases—were all without ascorbic acid for 10 or more days prior to tissue and blood analysis. In all but 1 of these 6 cases, the buffy layer ascorbic acid content was also subnormal. Whether the occurrence of 3 bleeding ulcers in this small group with high tissue and low plasma ascorbic acid content is simply the result of previous low intake of the vitamin or whether it is the result of unusual migration of ascorbic acid to the tissues as a response to either this disease or to stress requires further clarification.

Certain other tissues and organs made available at operation were analyzed, as well as the patient's blood and fascia, for their ascorbic acid content, and these data are tabulated in Table IV.

SUMMARY AND CONCLUSIONS

Twenty normal adults showed average ascorbic acid values of 15.2 milligrams per 100 grams of white cell-platelet layer, 0.73 milligram per cent of whole blood, and 0.69 milligram per cent of plasma.

Two hundred consecutive surgical patients had average ascorbic acid values of 14.5 milligrams per 100 grams of buffy layer, 0.48 milligram per cent of whole blood, and 0.36 milligram per cent of plasma.

In the seriously ill patient, the ascorbic acid content of tissues, buffy coat, whole blood, and plasma appears to be in a state of constant flux. The more or less predictable relationships between blood and tissue ascorbic acid seen in acute deprivation experiments in otherwise normal individuals are not found in the sick patient.

From studies of dietary histories and from blood and tissue levels, it was apparent in this series that anterior rectus fascia levels below 1.5 milligrams per 100 grams indicated significant ascorbic acid deficiency according to the Roe-Kuether method.

Ascorbic acid deficiency in the fascia of surgical patients is better reflected by plasma than by buffy layer levels.

Plasma ascorbic acid levels below 0.2 milligram per cent are indicative of tissue deficiency of ascorbic acid in surgical patients.

Providing no recent ascorbic acid has been given, plasma values above 0.2 milligram per cent are indicative of "adequate" tissue ascorbic acid, and plasma values above 0.4 milligram per cent are indicative of "adequate" tissue and buffy coat ascorbic acid, as measured by the patients' clinical response and associated blood and tissue levels in this series.

Among patients having a relatively low intake of ascorbic acid over long periods the tissue and plasma ascorbic acid may be low with a relatively high buffy layer level. The wounds of such patients show relatively good healing clinically.

When both tissue and buffy layer ascorbic acid content are low, clinical scurvy may become apparent. When they are both very low, poor healing may be evident.

Two patients having very low tissue and buffy coat ascorbic acid reacted very poorly on the operating table and died 72 and 24 hours after prostatectomy and ventral herniorrhaphy, respectively. The first patient showed only interstitial myocarditis with focal necrosis at autopsy; the second showed generalized oozing into his wound and cirrhosis of the liver. The clotting time in this latter case was normal.

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CONGENITAL MALFORMATIONS OF THE RECTUM AND ANUS

II. Associated Anomalies Encountered in a Series of 120 Cases

THOMAS C. MOORE, M.D., and EDWIN A. LAWRENCE, M.D., F.A.C.S.,
Indianapolis, Indiana

No study of congenital malformations of the rectum and anus can be complete without a careful consideration of the problems presented by the high incidence of associated congenital abnormalities. Although a tendency toward multiplicity in congenital anomalies has long been recognized, few anomalies are accompanied by other congenital difficulties so often. This frequent occurrence seriously compromises the overall prognosis, even in cases in which the anorectal problem has been satisfactorily dealt with, and has been largely responsible for close to one-half of the deaths in cases of anorectal malformation. Harken, in 1942, called attention to the gravity of the imperforate anus problem by pointing out the high nonsurgical mortality. Since recent advances in the field of pediatric surgery have brought many of these associated anomalies within the scope of increasingly effective surgical intervention, it is particularly important that this aspect of the management of anorectal anomalies be reviewed so that earlier recognition can be achieved and treatment carried out before irreparable damage has been done.

During the past 25 years, 120 cases of congenital malformations of the rectum and anus have been studied at this hospital (17), of which number 23 have been previously reported by Berman in 1938. Other congenital abnormalities were encountered in 86 of the 120 cases (72 per cent), a higher incidence than has been reported in other series (Table I). Furthermore, we suspect that the true incidence of accompanying malformations may even be somewhat in excess of 72 per cent since they were found in 33 of the 34 patients upon whom postmortem studies were carried out (97 per cent).

It was found that a number of infants will succumb to multiple congenital anomalies in the first few days of life and that others will die due to their presence before the age of 2 years. Sixty-three of the 120 patients (53 per cent) were admitted to the hospital during the first 4 days of life and only 13 (11 per cent) were first seen after 2 years of age. It is, therefore, of interest that as high as 44 per cent of the 165 patients of Mayo and Rice were found to have other malformations since only 18 of their patients (11 per cent) were seen during the first 48 hours of life and 87 (53 per cent) were not seen until after 2 years of age.

The generally accepted classification of anorectal anomalies described by Ladd and Gross (11) has been employed. In type 1 there is a stenotic narrowing of the rectum or anus. In type 2 the rectal pouch is quite low, while in type 3 several centimeters may separate the blind-ending rectal pouch from the anus. Rectal atresia occurs in type 4 with a normal appearing anus and lower rectum. The observed incidence of associated anomalies was somewhat greater in types 3 and 4 (Table II), perhaps in part due to the fact that the mortality, and, hence, the relative frequency of postmortem examination, was greater in these groups (Table III). We have not regarded congenital fistulous communications with the blind-ending rectal pouch as additional abnormalities since they are more properly classified as an integral part of the anorectal malformation.

The largest number of associated anomalies to be found in a single case was 6, and the

From the Department of Surgery, James Whitcomb Riley Hospital, the Indiana University Medical Center, Indianapolis, Indiana.

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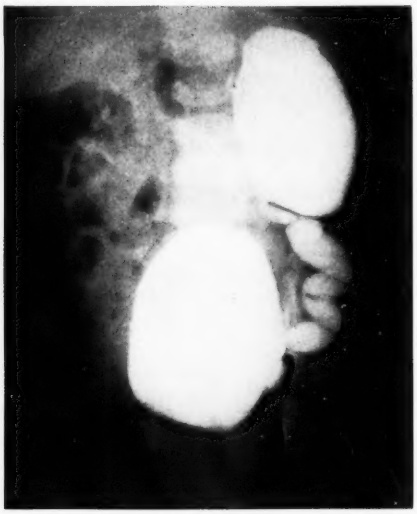

Fig. 1. Roentgenologic demonstration of left megaloureter and hydronephrosis by delayed reflux pyelography.

TABLE I.—REPORTED INCIDENCE OF ASSOCIATED ANOMALIES

Author	No. of cases	No. of cases with associated anomalies	Per cent with associated anomalies
Ladd and Gross (12)	214	60	28
Mayo and Rice	165	73	44
Bacon and Hering	98	29	30
Lu	74	16	22
Norris, Brophy, and Brayton	52	24	46
Crowell and Dulin	28	13	46
Lee	16	11	69

TABLE II.—INCIDENCE OF ASSOCIATED ANOMALIES ACCORDING TO TYPE OF ANORECTAL MALFORMATION

Type	No. of cases	No. of cases with associated anomalies	Per cent with associated anomalies
1	5	3	60
2	20	11	55
3	90	68	76
4	5	4	80
Total	120	86	72

TABLE III.—INCIDENCE OF ASSOCIATED ANOMALIES IN AUTOPSY CASES

Type	No. of cases	No. of cases with associated anomalies	Per cent with associated anomalies
1	1	1	100
2	3	2	67
3	28	28	100
4	2	2	100
Total	34	33	97

over-all total identified was 190 (Table IV). They were most frequently encountered in the urinary tract. Fifty-four urinary tract anomalies were discovered in 41 of the 120 cases (34 per cent). A possible explanation for this frequent occurrence may lie in the observation that a portion of the permanent renal system is derived as a dorsal outgrowth from the wolffian duct, near its termination in the cloaca, at approximately 1 month of fetal life. During this time, developmental aberrations are taking place in the cloacal and anal regions which give rise to the anorectal anomalies—and perhaps to urinary tract anomalies as well.

Megaloureter and hydronephrosis, in most instances presumably due to impaired function at the ureterovesical junction, were demonstrated in 18 cases by roentgenologic study, laparotomy, or postmortem examination as illustrated by the following 2 cases. Routine urinary tract evaluation studies, including reflux pyelography, were carried out recently on a 14 month old type 3 patient. They revealed the presence of a previously unsuspected left megaloureter and hydronephrosis (Fig. 1). There was no reflux of the radiopaque media up the right ureter. The patient died following colostomy closure and examination of the bladder at necropsy showed marked enlargement of the left ureteral orifice, the right ureteral orifice appearing normal (Fig. 2). The right ureter and kidney were of normal size with microscopic evidence of mild renal aplasia. The left ureter was considerably larger than normal and the aplastic left renal parenchyma was almost completely destroyed, remaining simply as the thin lining of a distended, hydronephrotic sac. Microscopic study of sections taken from the left ureterovesical junction demonstrated the presence of numerous autonomic ganglia in the adventitia of this area (Fig. 3).

TABLE IV.—ASSOCIATED ANOMALIES

	No. anomalies
Urinary tract	54
Megaloureter and hydronephrosis	18
Ureteropelvic obstruction	2
Absence of kidney	5
Absence of kidneys, bladder, and urethra	1
Ectopia of kidney	7
Horseshoe kidney	2
Multiple pelves and ureters	3
Incomplete rotation of kidney	2
Hypoplasia of the bladder	4
Bladder hypertrophy	1
Absence of external genitalia	1
Rudimentary penis	1
Hypospadias	7
Spinal-neurological	41
Myelomeningocele	2
Spina bifida occulta	7
Diastematomyelia	1
Other lumbar and sacral deformities	13
Pilonidal sinus	2
Rudimentary tail	1
Neurogenic bladder	3
Mental retardation	4
Hydrocephalus	4
Microcephaly	1
Mongolism	2
Cerebral hypoplasia	1
Cardiovascular	20
Congenital heart disease	19
Stenosis common iliac artery	1
Alimentary tract	27
Esophageal atresia with tracheo-esophageal fistula	7
Duodenal atresia	3
Hypoplastic pancreas	2
Absence of gallbladder	4
Absence of bile ducts	1
Malrotation of intestines	4
Meckel's diverticulum	6
Abdominal wall	15
Umbilical hernia	6
Omphalocele	2
Inguinal hernia	6
Hydrocele	1
Female genital tract	10
Bicornuate uterus	4
Septate vagina	4
Absence of uterus	1
Hydrometrocolpos	1
Extremities	10
Clubfoot	8
Supernumerary thumb	1
Fusion of lower extremities	1
Miscellaneous	13
Bifid scrotum	6
Harelip	1
Cleft palate	1
Deformed ears	1
Absence of ear	1
Deafness	1
Strabismus	1
Separation of pubic rami	1
Total	190

Fig. 2. Photograph of bladder obtained at necropsy showing abnormal enlargement of left ureteral orifice (*L.U.*).

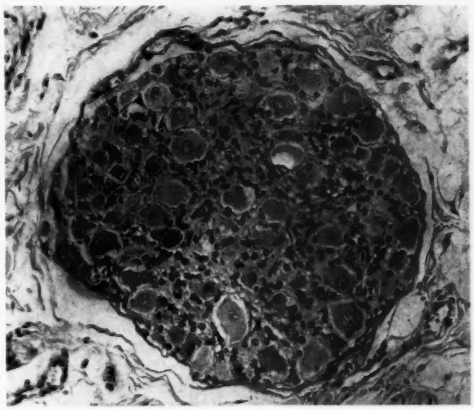

Fig. 3. Photomicrograph of one of the several normal appearing autonomic ganglia encountered in the adventitia of the left ureterovesical junction pictured in Figure 2.

Another patient, a 2 day old type 3 male, was found to have marked bilateral ureteral dilatation during abdominoperineal anoplasty. Both ureters were dilated, to approximately 2 centimeters in diameter, down to the ureterovesical junction. The bladder, which was not distended, was opened and there was no evidence of demonstrable mechanical obstruction at the bladder neck or in the region of either of the patent ureteral orifices. Suprapubic bladder drainage was maintained for 4½ months postoperatively. Intravenous urograms at 10 months of age showed persistent ureteral dilatation, although less marked than was seen at operation. Overdistention of the bladder with radiopaque media has failed to show reflux up either ureter.

These 2 cases illustrate some of the more perplexing aspects of the megaloureter problem as encountered in association with anorectal malformations. Although the pathological and physiological alterations leading to the development of megaloureter have not been clearly identified, most authors agree that they are probably neurogenic. The solution of this problem has been hampered by a lack of convincing evidence concerning the influence of both intrinsic and extrinsic autonomic innervation on ureterovesical junction function (7).

Caulk, in 1923, was one of the first to call attention to the function of the ureterovesical valve mechanism in the etiology of megaloureter and to point out the similarity of this

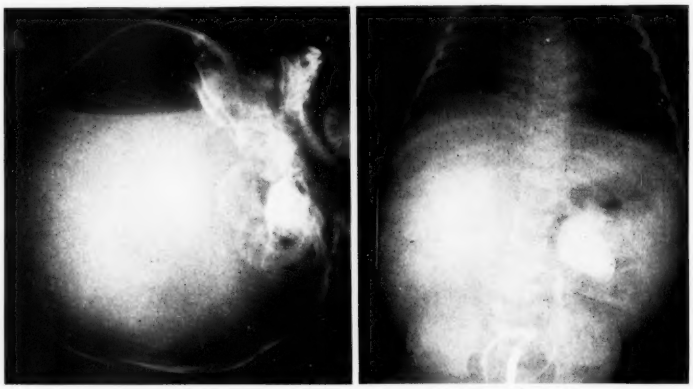

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Fig. 4. Roentgenograms carried out on a patient with type 2 imperforate anus showing evidence of (a) defective cerebral development, by subdural air injection, and (b) hydronephrosis from ureteropelvic obstruction, by retrograde pyelography.

condition to megacolon. Autonomic ganglia were shown by Muller, in 1924, to be normally quite numerous at the ureterovesical junction and present also in the bladder and extramural ureters, although in considerably smaller numbers. The suggestion was made in 1931 by Hurst and Gaymer-Jones that megaloureter was due to achalasia of the ureterovesical sphincter as the result of degenerative changes in the autonomic ganglia of the intramural segment of the ureter. The absence of autonomic ganglion cells in the intramural ureter in a case of megaloureter was reported in 1942 by Etzel. Swenson, MacMahon, Jaques, and Campbell have recently suggested that megaloureter, in some cases, may be due to bladder dysfunction resulting from a diminution in parasympathetic ganglion cells. The occurrence of megaloureters in association with the neurogenic bladder of paraplegia is well recognized (7) and it is possible that the basic lesion in some cases of megaloureter is located in the central nervous system rather than in the peripheral. Indeed, of the 2 reported "test" cases of Swenson and his associates, 1 patient was found to have moderately severe microgyria of the brain and the other was a congenital hydrocephalic. Two of the cases of megaloureter in our imperforate anus series were accompanied by demonstrable central nervous system pathology and in 1 other case there were multiple lumbar vertebral anomalies. Whether megaloureter is due to a nonneurogenic anatomic abnormality at the ureterovesical junction or to a deficiency of either intrinsic or extrinsic innervation remains to be established. It is through the continuation of such careful studies as Swenson and his group are carrying out that a solution to this difficult problem may ultimately be achieved. It is of interest that other urinary tract abnormalities were encountered in 8 of our 18 patients with megaloureter.

Due to the absence of a clear etiologic basis for megaloureter, the treatment of this condition is still empirical. Wangensteen (21) has reported the use of bilateral myotomy at the ureterovesical junction in a case of bilateral ureterectasis, with unilateral success. Swenson and his associates have obtained good results with prolonged suprapubic bladder drainage. Suprapubic bladder drainage was carried out in 3 of our cases with some improvement.

Of the other urinary tract abnormalities, exstrophy of the bladder was present in 4 patients, all of whom died. Two of these infants also had large omphaloceles. Death has resulted in a number of the cases from renal failure and the prognosis for several of the living patients is poor because of serious urinary tract disabilities. One type 3 patient, treated by perineal anoplasty, is living at 8 years of age with intermittent pyuria and renal insuf-

ciency. A left nephrectomy was carried out on this patient at 18 months of age for triplication of the renal pelvis with severe pyonephrosis and, at 3 years of age, a right ureteropelvic obstruction was treated operatively by division of an aberrant renal vessel. Another type patient, whose only treatment had been the creation of a colostomy at birth, is currently suffering from increasing renal insufficiency at years of age. A left flank exploration on this child has demonstrated the congenital absence the left kidney and ureter. In addition, his patient has megaloureter and hydronephosis on the right side, as well as a neurogenic bladder, for which a suprapubic cystostomy has been carried out.

Associated malformations frequently involved the vertebrae, particularly in the lumbosacral area, and the central nervous system. Vertebral anomalies, often multiple, were demonstrated in 18 patients. Evidence of cerebral deficiency was encountered in 12 cases. A similar tragic experience, in this regard, has recently been reported by Lanman in the management of duodenal atresia. He found 4 of the last 17 patients surviving operation at the Children's Hospital of Boston to be proved mongols, while 2 others were mentally deficient. One of our patients was shown to have a subdural hematoma complicating cerebral hypoplasia (Fig. 4a). This patient, who had survived the successful repair of a type 2 imperforate anus at birth and had tolerated a plastic procedure for the correction of a ureteropelvic junction obstruction (Fig. 4b) at 6 months of age, died of cardiac standstill on the operating table at 8 months of age while a bone flap was being turned in order to excise the subdural hematoma membrane.

As might be expected, anomalies were often encountered elsewhere in the alimentary tract. Other alimentary tract atresias were demonstrated in 10 cases. In 7 of the cases, there was an esophageal atresia with a tracheo-esophageal fistula (Fig. 5). The tracheo-esophageal fistula communicated with the distal esophageal segment in all but 1 of the cases. The anorectal anomaly was of type 2 in 1 case and of type 3 in 6 cases. Two infants, early in the series, died without any attempt at surgical correction of the esophageal atresia having been made. Another infant died when an effort at esophageal repair was abandoned for technical reasons. The last 4 patients, however, survived both anal and esophageal operative procedures. Ligation and division of the tracheo-esophageal fistula followed by end-to-end esophageal anastomosis was utilized in all 4. Although 1 patient died at 6 months of age of infectious diarrhea, the other 3 are living and doing well. A combined abdominoperineal anoplasty had been performed on 1 of these 24 hours after esophageal repair.

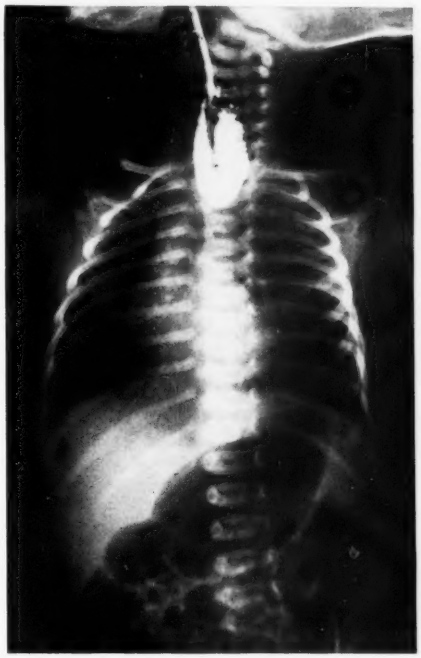

Fig. 5. Roentgenologic confirmation of esophageal atresia, in a patient with type 3 imperforate anus, by lipiodol instillation into the upper esophagus. The tracheo-esophageal fistula communicated with the distal esophageal segment.

Two of the 3 patients with duodenal atresia were premature. Both infants weighed slightly less than 3 pounds and died shortly after being admitted to the hospital. One patient survived gastrojejunostomy, following perineal anoplasty, and died at 6 months of age of multiple pulmonary abscesses accompanying a severe otitis media.

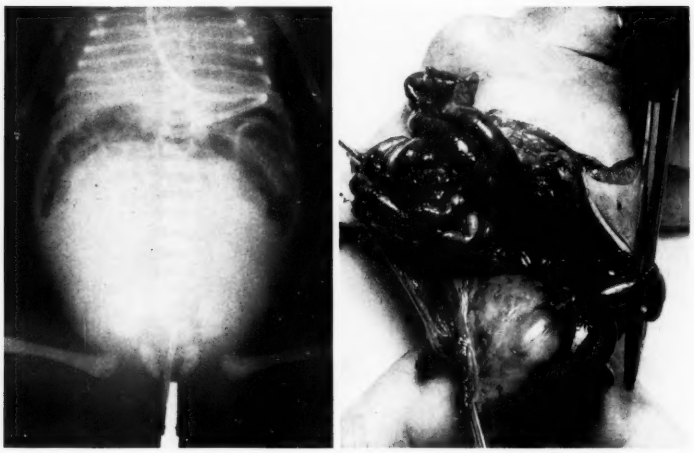

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Fig. 6. Demonstration of marked distention of hydrometrocolpos, into which a rectovaginal fistula opened, by (a) roentgenograms taken before suprapubic aspiration of 500 cubic centimeters of mucoid fluid and (b) postmortem examination. The presence of the tip of the catheter in the stomach (a) rules out the existence of esophageal atresia.

Congenital heart disease was found in 19 cases. Although the precise nature of the cardiac or great vessel abnormality was not determined in many of them, postmortem examination in a few demonstrated anomalies now known to lie within the scope of effective cardiac surgery (3). It is also possible that other cardiac anomalies, such as the large auricular septal defects which were encountered at necropsy in 2 cases, may some day be amenable to operative correction. Congenital stenosis of the left common iliac artery was present in 1 case, possibly due to "excessive obliteration" of the fetal umbilical artery.

Congenital hernias occurred often. Both cases with a large omphalocele were further complicated by the existence of an exstrophy of the bladder. An attempt was made to carry out the Gross operation (8) on 1 of these infants. It was only partially successful due to the inadequacy of the available skin flaps and the patient died 8 days postoperatively. Clubfoot was the most frequently encountered extremity anomaly, being found in 8 cases.

Unusual combinations of anomalies were found in several cases. One cyanotic female infant with a type 3 imperforate anus was admitted to the hospital at 12 hours of age in severe respiratory distress. Her abdomen was quite distended. Roentgenograms demonstrated marked diaphragmatic compression from a large intra-abdominal mass (Fig. 6a). Although 500 cubic centimeters of yellowish mucoid fluid were removed by suprapubic needle aspiration, the infant died 3 hours after admission. Postmortem examination revealed a rectovaginal fistula entering a hydrometrocolpos (Fig. 6b), the distention of which had been the cause of the mechanical interference with diaphragmatic activity. In addition, there was a bicornuate uterus, a partially septate vagina, absence of the left kidney and ureter, and absence of the gallbladder and bile ducts. Another infant, several weeks premature and weighing 3 pounds, was admitted to the hospital at 16 hours of age with a type 3 imperforate anus, absence of the external genitalia, a rudimentary tail, fusion of the lower extremities, and bilateral clubfoot (Fig. 7). The urinary bladder could not be found on exploratory laparotomy and a colostomy was performed. The infant died 36 hours after admission and was shown, at necropsy, to have an absence of the kidneys, ureters, bladder, and urethra. Microscopic examination of

intra-abdominal testes established the sex of this infant. Although prematurity was occasionally associated with multiple anomalies, it occurred in only 8 of the 120 cases in this series.

DISCUSSION

The incidence of associated anomalies in cases of anorectal malformation is high. Twenty-three of the 50 deaths in this series have been due, in large part, to their presence (17). Furthermore, the prognosis for several of the living patients must be regarded as poor because of the existence of additional congenital defects, principally those involving the urinary tract. It is, therefore, important that the early recognition and treatment of these associated anomalies be considered as an integral part of the management of the imperforate anus "syndrome."

Although the grave nature of some of the associated malformations, or combinations of them, is at times virtually insurmountable, the presence of remediable anomalies should be anticipated so that they may be dealt with before irretrievable injury has occurred. The recent results with esophageal atresia have been most heartening in this regard. Esophageal atresia is one of the few important anomalies which generally require treatment prior to the relief of the intestinal obstruction from anorectal imperforation. It is, therefore, the policy at this institution to pass a soft rubber catheter through the esophagus into the stomach in all imperforate anus patients at the time of their admission to the hospital and to confirm the presence of the tip of the catheter in the stomach when the initial Wangensteen-Rice roentgenograms (22) in the upside-down position are taken.

The presence of other alimentary tract atresias should also be ruled out. Abdominoperineal anoplasty is now being used at this hospital in cases of types 3 and 4 imperforate anus and the recognition of other intra-abdominal abnormalities is found to be among its many advantages.

Due to the high incidence of associated urinary tract anomalies, we feel that careful urinary tract studies should be carried out on all patients during the first few months of life and at intervals thereafter; so that appropriate treatment may be carried out before severe renal damage and insufficiency occur.

Fig. 7. Photograph of a premature type 3 male showing a rudimentary tail, fusion of the lower extremities, and the absence of an anal opening.

Roentgenologic studies of the lumbosacral spine may reveal unsuspected vertebral anomalies. Periodic psychometric and neurological examinations may also be helpful in following the development of these patients.

In view of current rapid advances in the field of cardiac surgery, it is especially important that no congenital anomalies of heart or great vessels, amenable to surgery, be overlooked.

SUMMARY

1. Additional congenital abnormalities were encountered in 86 of the 120 cases of congenital malformations of the rectum and anus (72 per cent) and in 33 of the 34 patients upon whom careful postmortem studies were carried out (97 per cent).

2. One hundred and ninety associated anomalies were found and are tabulated according to organ systems involved.

3. Associated anomalies occurred most frequently in the urinary tract. Fifty-four urinary tract anomalies, excluding congenital rectourinary tract fistulas, were found in 41 of the 120 patients (34 per cent). The incidence of megaloureter in this series (18 cases) is reported and the question of its etiology and treatment is discussed.

4. Vertebral anomalies, commonest in the lumbar and sacral regions, were frequently multiple and were identified in 18 cases.

5. Evidence of cerebral deficits in 12 cases suggests that one must anticipate mental retardation in from 10 to 20 per cent of the surviving patients.

6. Atresias elsewhere in the alimentary tract were found in 10 cases, 7 of them occurring in the esophagus with tracheo-esophageal fistula.

7. Nearly one-half of the deaths in 120 cases of anorectal anomalies were due to the presence of other congenital malformations and the prognosis for a number of the living patients is poor because of them.

8. The importance of the early recognition and treatment of these associated anomalies as an integral part of the imperforate anus "syndrome" is stressed.

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RESULTS OF TREATMENT OF FRACTURED MANDIBLES BY THE ANDERSON SPLINT

M. STEIN, M.B.B.Ch.(Wits.), F.R.C.S. (Edin.), D.A., D.G.O.,
M. N. TEUBES, M.B.B.S.(Lond.), F.R.C.S. (Edin.), and
S. SHER, M.B.B.Ch.(Wits.), Johannesburg, South Africa

CORONATION Hospital is one of the non-European or Bantu units of the Johannesburg General Hospital. Over 60,000 new cases are seen at this hospital each year, and of these over 3,000 traumatic cases are actually admitted. Crimes of violence are extremely common among the Bantu, and an opportunity arose for analyzing the cases of fractured mandibles during the period 1948 to 1950. Anderson splints were used in the treatment of 125 of these.

Most of these cases were compound into the mouth, but only a few were compound externally. This was probably due to the type of injury, which in the majority of cases, was caused by a blow on the jaw with fist, stick, boot, or brick. Occasionally shock, skull, or brain damage was present as an associated injury, but surprisingly, this was not a prominent feature in our series of cases. Often, however, a history of concussion was obtained, though postconcussional sequelae were extremely rare.

Approximately 20 per cent of the cases did not require any immobilization other than perhaps a bandage. The following are examples of fractures that may be treated conservatively:

1. *Neck of condyle.* When these are single fractures conservative treatment will give a good result, from the functional point of view, provided the head is not dislocated through the capsule (Fig. 1).

2. *Ascending ramus.* This type of fracture is extremely rare. The fracture line either runs transversely across the ramus immediately above its junction with the horizontal ramus, or vertically from the angle to the mandibular notch. There is little or no separation and the fracture is adequately splinted by the muscles of mastication.

3. *Angular fractures.* Only certain selected fractures in this area should be treated conservatively. The following are some of the criteria which are considered favorable in assessing this fracture for this line of treatment: (a) The fracture must be simple. (b) There must be no pain at the fracture site when the mouth is gently opened and closed. (c) There must be no displacement. It should be noted that displacement depends largely on the direction of the fracture line, and thus clinical examination is essential, in contrast to the dangerous assessment of x-ray plates alone. (d) When it is considered that the fracture line passes through the attachments of the masseter and internal pterygoid muscles, and that these muscles will tend to oppose the fractured surfaces.

4. *Fractures of the horizontal ramus.* A crack fracture can occasionally be treated conservatively, provided that there are no infected teeth and that the patient is trustworthy. However, one patient in the series was considered to have sustained a crack fracture, and was consequently treated conservatively. Two weeks later it became necessary to incise an abscess at the fracture site! In this case movement had caused a simple fracture to become compound into the mouth.

The clinical test of "springing" is perhaps the most important criterion, but too much caution cannot be exercised.

TECHNIQUE OF SPLINTAGE

Preoperative care. In most cases it is advisable to delay the application of the Anderson splint for a few days. During this period there is usually considerable swelling of the jaw, which, besides increasing the difficulty of

From the Department of Surgery, University of Witwatersrand, Johannesburg.

Fig. 1.

Fig. 2.

Fig. 1. Fracture of the neck of condyle.
Fig. 2. Fracture of the angle of the mandible treated with Anderson splint.

the operation, must be considered as latent infected tissue. Advantage is taken of this period to start a "penicillin umbrella" and to improve the hygiene of the mouth with antiseptic mouth washes. Meanwhile the mandible should be splinted to the maxilla with the aid of a "barrel" bandage. When applying the bandage, care should be taken to see that the mandible is drawn vertically upward against the maxilla. In double fractures a carelessly applied bandage may push the mobile fragment backward.

Anesthesia. The average patient is prepared with atropine, grain 1/75, three quarters of an hour before going to the theater. Omnopon is not used as it depresses the respiratory center and cough reflex, and it is desirable to have the patient awake on completion of the operation. On arrival of the patient in the anesthetic room, the nostrils are sprayed with a solution of 2 per cent decicaine with adrenalin. Then a short acting muscular relaxant (1 to 2 c.c.) is given intravenously, and this is followed immediately through the same needle by sodium pentothal (2½ per cent solution) until the patient is asleep. The patient is then intubated through the nose by direct laryngoscopy. The mouth and throat are cleared by suction, the throat packed off. Anesthesia is maintained by cyclopropane and oxygen.

Technique. The line of fracture is traced upon the overlying skin. A point is chosen

Fig. 3.

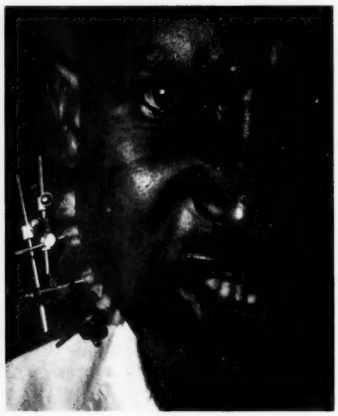


Fig. 4.

Figs. 3 and 4. Photographs showing splints in position. Note the exaggerated protrusive bite in Figure 4.

about half an inch from the fracture, and a pin, attached to a hand drill, is punctured through the skin until bone is reached. During the insertion of the pins the facial artery and the mental nerve must be avoided. The inferior dental canal and the roots of the teeth can be avoided by studying the x-ray films. Beyond the mandible the carotid vessels and the lingual artery and nerve may be in danger. By feeling with the point of the pin a spot about a quarter of an inch from the inferior border of the mandible is selected for the insertion of the pin. An assistant applies counterpressure by inserting a finger inside the mouth. Steadily and firmly the pin is drilled into the bone at an angle of 60 degrees. When the inner cortex grips the thread it is the signal that 1 or perhaps 2 more turns will be sufficient. The second pin is then inserted about 1 inch from the first.

A single clamp is now passed over each pin and a connection rod, with a double clamp in the middle, is passed through the single clamps. The pins are gently drawn toward each other, and the nuts on the single clamps are then tightened. This is called a "fracture unit." A similar fracture unit is applied to the mandible on the other side of the fracture line. The 2 fracture units are joined by a connecting rod passing through the 2 double clamps. The double clamp may have to be applied at one or the other end of the unit and not in between the pins, to facilitate the application of the

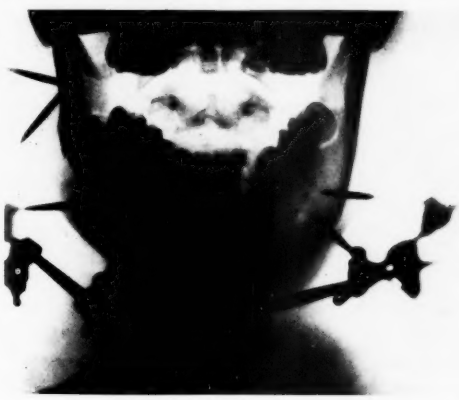


Fig. 5. A roentgenogram taken with the mouth widely opened.

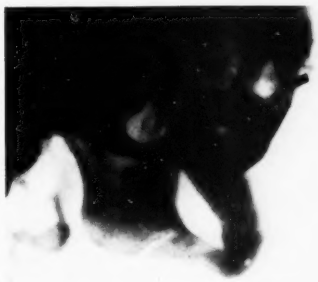


Fig. 6. The edentulous mandible.

connecting rod. In double fractures, 3 fracture units must be inserted, and these joined together by means of 2 connecting rods.

Occlusion and accurate alignment of the fractured mandible are now obtained, and the double clamps tightened. Vaseline gauze dressings are put around the base of each pin.

Any teeth which require extraction are then removed. Tooth extraction was not performed as a routine measure in every case, but only if the tooth was broken, loose, or infected. Only 30 per cent of the patients had teeth extracted, and there does not appear to be any cause to regret this conservative attitude. It is important, however, that infected teeth in the neighborhood, although not actually in the fracture line, can cause local infection. However, the position must be carefully considered, as a difficult extraction in a fractured mandible may cause further injury to blood vessels and nerves, and the persistent search for a broken off root may be a potent cause of osteomyelitis.

Only very rarely is intermaxillary fixation required in addition to external splintage. The following day the patient is examined and if occlusion is found to be unsatisfactory, the fragments are remanipulated under intravenous omnopon which allows the co-operation of the patient. Remanipulation had to be performed in 22 (17.6 per cent) of the cases in this series.

After approximately 6 weeks the pins are extracted under morphine sedation. Occasionally a parotid fistula may be troublesome after the pins are removed, but this usually disappears within the first few days.

Considering the type of injury, it is not surprising that 88 per cent of the cases occurred in males, and that about 50 per cent of the cases occurred in the age period of from 20 to 30 years. However, it is surprising that one third of the patients did not seek treatment on the first day of injury. In fact, their average day of attendance was 7 days after injury.

CLASSIFICATION OF FRACTURES

Mandibular fractures may be classified as: (a) simple or compound, (b) single, double, triple or comminuted, or (c) crack, mobile or impacted.

The site of fracture may be at: (1) the horizontal ramus, (2) the angle (posterior to a vertical line dropped from the last molar tooth), (3) the neck of the condyle, (4) the symphysis menti, (5) the ascending ramus, and (6) the coronoid process.

Sixty-three per cent of the cases were single fractures, and most of these (92 per cent) occurred either at the angle or in the horizontal ramus in about equal frequency. Only 6 per cent had fractures in the region of the neck of the condyle. Double fractures occurred commonly at the angle and horizontal ramus, or at both horizontal rami. Only very occasionally is the neck of the condyle the second fracture.

The results were good in 95 per cent of the cases treated at Coronation Hospital. The 5 per cent that gave unsatisfactory results were difficult cases with multiple fragments, and perhaps the results would have been the same whatever the method of treatment. In addition all cases in this group were notable in that the patients showed considerable delay before seeking treatment at the hospital.

The argument used most frequently against the use of the Anderson splint is that it causes infection of the bone. However, no case can be recalled in which an osteitis occurred at the site of insertion of a pin, and none occurred in this series of 125 cases. There were 11 cases (8.8 per cent) of superficial pin track infection, which did not result in loosening of the pins and cleared up as soon as the pins were removed. Among the whole series there were 18 cases (8.6 per cent) of infection of the hematoma around the fracture site and 3 cases of local periosteal reaction. The infected hematoma requires early drainage, preferably externally, and intensive chemotherapy.

Bony union usually occurs in the second half of the first year. Before this time, although there is only fibrous union, the functional result is sound enough for almost normal mastication. Poor immobilization and infection will delay bony union. Poor alignment will defeat the primary object of treatment of the restoration of normal function of the lower jaw. Thus it is stressed that there should be no delay in remanipulating the fracture to obtain perfect occlusion.

ADVANTAGES OF THE ANDERSON SPLINT

1. The splint is simple to apply. The whole operation is usually completed within 30 minutes.
2. The Anderson splint allows adequate immobilization with the advantage that neither feeding nor vomiting is a problem at all (Fig. 5).
3. The mouth is kept much cleaner. This allays infection at the fracture site.
4. There is no residual stiffness of the temporomandibular joint after removal of the splints.
5. No unsightly scars will result.
6. Teeth may be unsuitable for wiring.
7. It is one of the few methods which are applicable to the edentulous mandible (Fig. 6).
8. It adequately immobilizes the posterior edentulous fragments in angular fractures or those in which the last molars have been removed.
9. It is ideal when there is an associated fracture of the maxilla.
10. The patient is incomparably more comfortable.
11. It can be used in nearly all cases of fractures of the mandible, except in children, where it may harm the permanent tooth follicles.

CONGENITAL HERNIA OF THE DIAPHRAGM WITH SPECIAL REFERENCE TO RIGHT-SIDED HERNIA OF THE LIVER AND INTESTINES

ERNEST E. ARNHEIM, M.D., F.A.C.S., New York, New York

THE surgical pathology and treatment of the most common types of congenital diaphragmatic hernia in infancy and childhood will be considered in this communication, followed by an analysis of personal and reported cases in which defects of the right side of the diaphragm resulted in herniation of the liver and intestines into the thorax. The clinical manifestations and roentgenologic observations will be described in the case reports.

PATHOLOGY

Failure of fusion of the various developmental portions of the diaphragm or failure of muscular development in these regions result in congenital hernia of the diaphragm (Fig. 1). The most common site of developmental abnormality of the diaphragm is in its posterior portion. The identity of location and relations of the pleuroperitoneal opening in the fetus and the hernial opening suggest that a persistent pleuroperitoneal hiatus is the developmental cause. The location of this defect in the costovertebral area has become associated with the name "foramen of Bochdalek." Not infrequently the posterior defect in the diaphragm is larger and is thought to result from failure of formation of that portion of the diaphragm derived from the pleuroperitoneal membrane; it occupies the posterolateral portion of the diaphragm and usually extends posteriorly from the eighth rib and medially to the esophageal hiatus.

These types of hernias are much more common on the left side than on the right side. From an embryologic study this is explained by the earlier closure of the right pleuroperitoneal membrane and the protection of the liver.

From the Surgical and Pediatric Services of the Mount Sinai Hospital, New York, New York.

Most congenital diaphragmatic hernias do not have a hernial sac and there is a free communication between a pleural cavity and the abdomen ("false" hernia), but if herniation occurs at a slightly later stage of development when pleural and peritoneal membranes have closed the defect, but with incomplete muscular formation, a sac of peritoneum and pleura is present ("true" hernia). The sac is a thin membrane which is usually attached to a cresent-shaped shelf of diaphragm laterally, a segment of diaphragm medially, and is reflected over the viscera in the thorax as a hood.

The most common abdominal viscera involved in congenital diaphragmatic hernia are the small intestines, and the ascending and proximal transverse colon, i.e., that portion of the intestinal tract derived from the midgut in the fetus. Occasionally the stomach is herniated, and either the spleen or liver according to the side of the defect. It is thought that patency of the pleuroperitoneal foramina is prolonged since the return of the intestinal loops from the umbilical sac to the peritoneal cavity is usually from 2 to 3 weeks after the pleuroperitoneal foramina are closed (Fig. 2). At first inspection the abdominal viscera seem to be arranged in a confused fashion, but it should be recalled that the intestinal loops may be found in various stages of rotation in the thorax. There is a rudimentary attachment of the mesentery of the small intestine just below the superior mesenteric artery due to failure of the mesentery to become attached to the posterior abdominal wall along the oblique line low in the abdomen. There is also atypical fusion with the posterior body wall of the ascending colon. These changes indicate the probability that the intestines were already displaced in the fourth month of fetal life when the fusions usually occur. It is re-

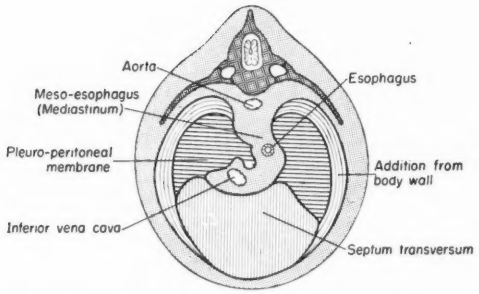


Fig. 1. Diagram indicating embryologic origin of various regions of the diaphragm.

markable that despite these displaced attachments fusion with the pleura does not occur, and adhesions between the herniated viscera and the intrathoracic surfaces rarely are found.

The displaced viscera in the thorax compress the homolateral lung which is usually found as an airless mass of dark red tissue compressed against the apex of the chest. The heart and mediastinal structures are displaced to the opposite side. The small size of the peritoneal cavity results from the absence of the herniated viscera.

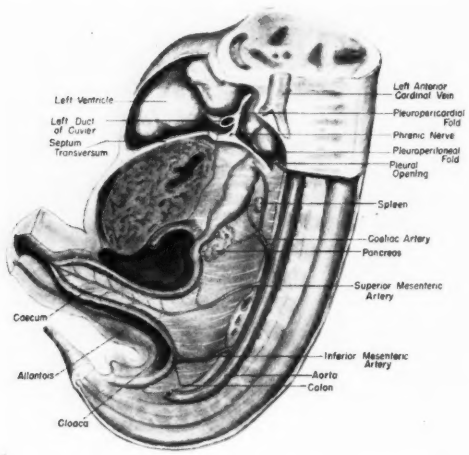


Fig. 2. Diagram of lateral dissection in an embryo of about 6 weeks, showing arrangement of the viscera and relations of the septum transversum and pleuropericardial and pleuroperitoneal folds. (Redrawn from Patten. Courtesy, The Blakiston Co.)

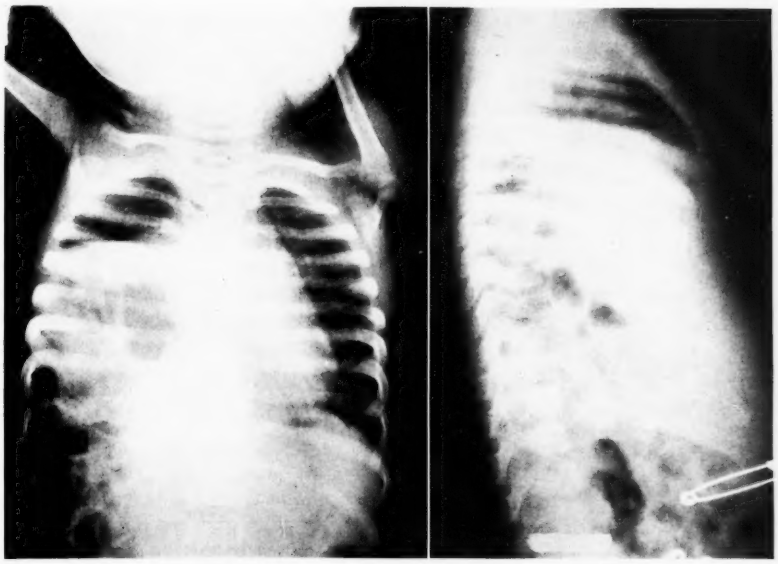
TREATMENT

Prompt surgical treatment of congenital diaphragmatic hernia is extremely important in the newborn period. A review of 30 newborn infants with this condition (Greenwald and Steiner) showed the duration of life to be less than 1 hour in 13, less than 24 hours in 9, and less than 36 hours in 2 patients. A waiting policy has resulted in many deaths in this age group, and too often the operation is withheld until the infant is in extremis. Improvements in anesthesia allow these infants to tolerate operation well. An added advantage of operation in the newborn period is the lack of distention of the intestines with air. The necessity for prompt operation continues during the first few weeks of life. A review of 210 cases of congenital diaphragmatic hernia (Hedblom) revealed that without operation 158 patients (75 per cent) died before the end of the first month after birth. Even if the infant survives the early age period the dangers of sudden respiratory or cardiac decompensation and intestinal obstruction remain. In an analysis of 68 operations for diaphragmatic hernia in children under 10 years of age (Hartzell) the operative mortality was 25 per cent in patients without intestinal obstruction, and 66.6 per cent in those with intestinal obstruction.

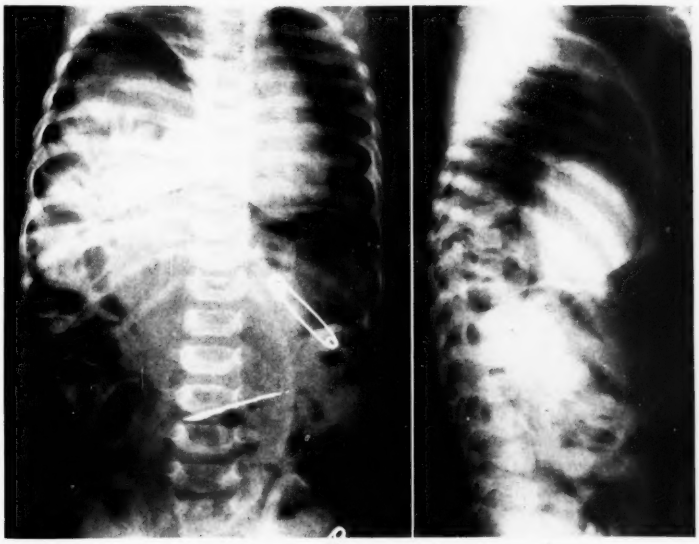
Before transportation to the operating room, inhalation of oxygen should be supplied through an incubator or cubicle, the stomach emptied through a Levin tube, and the necessary fluids administered through a cut-down intravenous in an ankle vein. The latter is utilized for transfusion of blood during operation.

The importance of anesthesia in determining the success or failure of operation is stressed by most surgeons writing on this subject. Positive pressure anesthesia is of the utmost importance and is given through a small closed system using a tightly fitting face mask.

The technique of reduction of the herniated viscera has been well described (Ladd and Gross, and Donovan), and is outlined in the case reports. The closure of small defects in the diaphragm presents few problems, but difficulties arise when the opening in the dia-



a Fig. 3. b



a Fig. 4. b

Fig. 3. Roentgenograms of the chest and upper abdomen (no contrast medium, Case 1). a, Dorsoventral projection, showing gas filled loops of intestine extending from upper abdomen into right side of thorax, absence of lateral portion of liver shadow, density in lower mediastinum, and lack of visualization of right side of the diaphragm. b, Lateral projection, showing loops of intestine situated posteriorly and a large opacity anteriorly in the thorax.

Fig. 4. Roentgenograms of the chest and abdomen after pneumoperitoneum (Case 1). a, Dorsoventral projection, showing the right pleural cavity occupied by the right lobe of the liver and gas filled loops of intestine separated from the hernial sac by air. b, Lateral projection, showing anterior position of the right lobe of the liver.

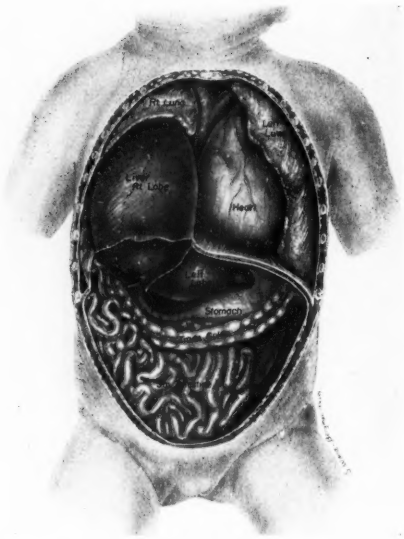


Fig. 5. Drawing illustrating the findings at operation in Case 1.

phragm is large, because in these cases there may be no attachment of the diaphragm to the posterior thoracic wall. A variety of procedures has been devised to close these large defects. The one I have found most helpful is making use of the renal fascia covering the anterior surface of the kidney and adrenal gland, according to a technique described by Weinberg, and described in the case reports. Donovan used a row of silk sutures with 2 needles on each, passing them through the diaphragm to the outside of the chest wall. Using a thoracic approach, Bettman and Bird fractured the ribs adjacent to the defect and pressed them inward to approximate the thoracic wall and the edges of the hernial ring. Harrington sutured the posterior margin of the diaphragm to the intercostal muscles with fascia lata, or advised shortening the diameter of the diaphragm by permanent interruption of the phrenic nerve or extrapleural rib resection. Belsey and Apley successfully employed the following procedure in 2 cases of right-sided congenital diaphragmatic hernia with large posterior defects: A thoracic approach was made through the bed of the ninth rib. After replacement of the abdominal viscera, the remnants of the diaphragm were freed from their attachments to the costal margin all around, and left with only their mediastinal origin and corresponding blood and nerve supply intact. The diaphragmatic defect was then closed by 2 rows of interrupted sutures of stainless steel wire and an overlap. The repaired diaphragm, much reduced in size, was reattached to the thoracic wall at a level 2 interspaces higher than normal with interrupted sutures of the same material.

In cases of "true" diaphragmatic hernia the operative procedure is modified to deal with the hernial sac after emptying it of its contents. Ladd and Gross advised a small incision in the sac to allow air to enter the pleural cavity, delivery of the sac into the abdomen, excision of the sac, and closure of the defect in the diaphragm. In 2 of my cases approximation of the large defect in the diaphragm was not possible, and the sac was utilized in the construction of a diaphragm (see case reports). This was accomplished by pulling the sac down to the level of the margins of the diaphragmatic attachments, with or without excision of a portion of the sac, and plication of the sac with superimposed rows of interrupted mattress sutures of silk. Although lacking the musculature of a normal diaphragm, follow-up examinations revealed a satisfactorily functioning structure. Truesdale employed plication of the sac in 1 case, followed by attachment to the thoracic wall. A similar technique was used in the repair of congenital eventration of the diaphragm by Bisgard, Butsch and Leahy, and Shellito and Barlett.

CASE REPORTS

CASE 1. A. K., a female infant, aged 10 months, was admitted to the Mount Sinai Hospital on April 6, 1945. The infant was born at full term of an uncomplicated pregnancy. The mother, father, and 4 siblings were living and well.

Except for a slow gain in weight, there were no complaints until 1 week before admission to the hospital when there was a sudden onset of fever, cyanosis, and dyspnea. These symptoms were partially relieved by the administration of oxygen and penicillin.

Physical examination revealed a poorly nourished infant in slight respiratory distress. The temperature was 100 degrees F., and the respirations 34. She weighed 12 pounds 11 ounces. There was no cyanosis. There were no abnormalities of the head, neck,

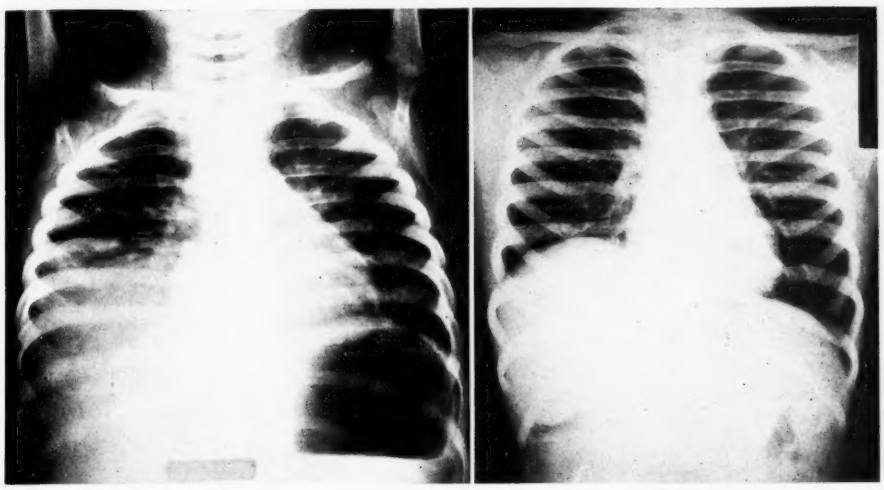


Fig. 6. Fig. 7.

Fig. 6. Roentgenogram of the chest taken 17 days after the operation (Case 1), showing an area of density in the lower right side, and absence of intestinal loops in the thorax.

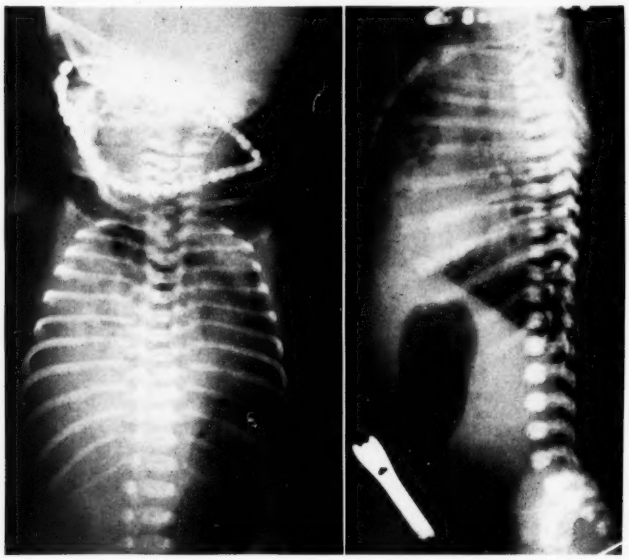
Fig. 7. Roentgenogram of the chest and upper abdomen taken 6 years and 7 months after the operation (Case 1), showing slight elevation of the right leaf of the diaphragm, satisfactory expansion of the lungs, and the right lobe of the liver in the abdomen.

extremities, or genitalia. Examination of the chest showed poor expansion of the right side. In the lower half of the right side there was marked dullness on percussion anteriorly and tympany posteriorly, absent breath sounds, and occasional bowel sounds were heard. The heart was normal and the liver and spleen were not palpable on abdominal examination.

Urinalysis revealed no abnormalities. The blood count showed a slight leucocytosis.

Plain roentgenograms of the chest and upper abdomen in the dorsoventral and right lateral positions, on the fourth day (Fig. 3), revealed dilated, gas-filled loops of intestine extending from the upper abdomen into the posterior part of the right side of the thorax to the level of the fourth rib; an absence of liver opacity was noted in the lateral portion of the right upper quadrant of the abdomen; a large opacity was seen anteriorly in the cardiac region. The roentgenologic findings were those of either hernia or eventration of the right diaphragm. Roentgenograms of the chest and abdomen after a barium meal, on the seventh day, revealed the stomach and small intestine to be located in the abdomen, and the cecum and ascending colon in the posterior portion of the right side of the thorax. Roentgenograms of the chest and abdomen in the dorsoventral and right lateral positions, after injection of oxygen into the peritoneum, on the ninth day (Fig. 4), revealed air passing into the right side of the thorax to the level of the third rib posteriorly, the roof formed by a thin membrane at this level; the right lobe of the liver was visualized anteriorly and loops of intestine posteriorly in the right side of the thorax. The membrane was thought to represent either the roof of a hernial sac or a high diaphragm.

Operation was performed on April 21, 1945, 15 days after admission. With the patient under ether anesthesia, a right oblique subcostal incision was made. Loops of small intestine were visible upon opening the peritoneal cavity. The stomach and left lobe of the liver were drawn over the right of their normal positions. The right lobe of the liver was not visible in the peritoneal cavity. After packing away loops of small intestine, a portion of the colon was found to pass upward into the right side of the chest along the posterior abdominal wall. A crescent-shaped shelf of the right diaphragm was visualized laterally and anteriorly. A catheter was passed upward into the thoracic cavity alongside of the colon. The operative findings are illustrated in Figure 5. The cecum, ascending colon, and hepatic flexure of the colon, which were suspended on a long mesenteric attachment, were withdrawn from the chest. The right lobe of the liver was then visualized in the right side of the thorax. The herniated portion of the liver was mobilized and brought down into the abdomen. A large space, about 10 centimeters in diameter, could be seen in the right side of the thorax, covered by a thin membrane near the apex of the chest. The right lobe of the liver was held in the abdomen, and the sac was pulled down to the levels of the margins of the diaphragmatic attachments. Plication of the sac was performed with superimposed rows of inter-



a b

Fig. 8. Roentgenograms of the chest and abdomen (without the use of a contrast medium, Case 2). a, Dorsoventral projection, showing an opacity with areas of translucency filling most of the right side of the thorax and the left side of the mediastinum, displacement of the heart to the left side, lack of visualization of the right side of the diaphragm, and absence of intestinal loops in the abdomen. b, Lateral projection, showing a large, rounded density in the lower half of the thorax with areas of translucency above and posterior to it, and an absence of intestinal loops in the abdomen.

rupted mattress sutures of silk. As the narrow edge of the diaphragm was approached laterally, the neighboring sutures were passed through it. At the termination of operation, the right lobe of the liver remained in place in the abdomen. The previously herniated portions of the colon were replaced in their normal anatomical positions in the abdomen. The peritoneum was closed with continuous sutures of chromic catgut, and the musculature, fascia, and skin with interrupted sutures of silk.

A transfusion of 250 cubic centimeters of citrated blood was administered during operation. At the end of operation the pulse was 140 and the respirations 60 and shallow. After operation, the indwelling Levin tube was attached to a suction apparatus. The infant was placed in an oxygen cubicle, and penicillin was started.

During the first postoperative day the highest temperature was 103 degrees F.; the pulse varied from 140 to 160, and the respirations from 60 to 80. There was moderate abdominal distention and bile-stained drainage from the gastric tube. There was no dyspnea or cyanosis. On the second day fluid feedings were retained, the bowels moved, and gastric siphonage was discontinued. The temperature, pulse, and respiratory rates remained elevated. A transfusion of 125 cubic centimeters of citrated blood was given. During the remainder of the first postoperative week the temperature, pulse, and respiratory rates were normal. Feedings were well taken, and there were no abnormalities of bowel function. The infant was removed from the oxygen cubicle on the fifth day. Penicillin was discontinued on the seventh day. The wound healed by primary union, and the skin sutures were removed on the seventh day.

During the second and third postoperative weeks the infant remained well, and there was a progressive gain in weight. The temperature, pulse, and respiratory rates remained normal. Examination of the chest and abdomen showed no abnormalities, except for slight dullness to percussion in the lower right side of the thorax. A roentgenogram of the chest, on the seventeenth day (Fig. 6), revealed an area of density on the right side extending to the level of the sixth rib posteriorly. The infant was discharged in excellent condition on the twenty-fourth postoperative day, weighing 13 pounds 5 ounces.

Follow-up examinations were made at 6 month intervals for 5 years and 9 months after operation. All of these examinations revealed progressive mental and physical development, and the child had

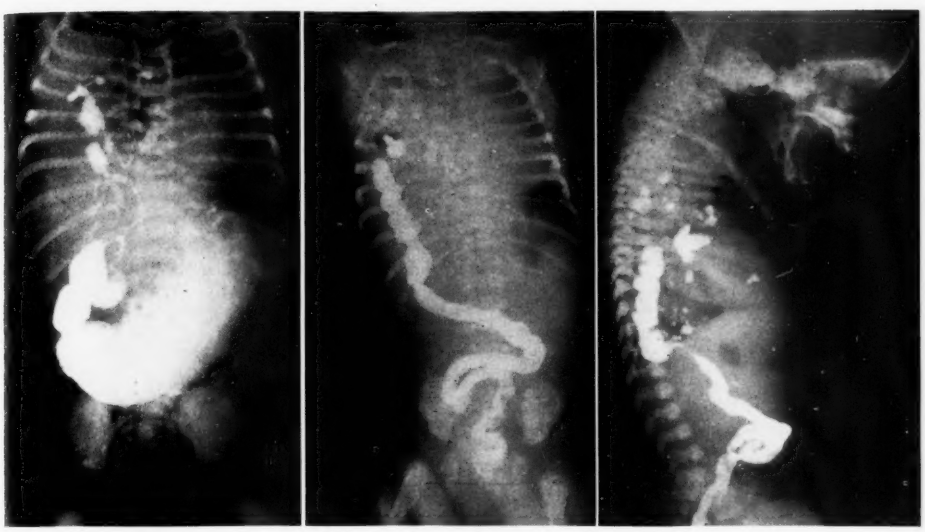


Fig. 9. a Fig. 10. b

Fig. 9. Roentgenogram of the chest and abdomen after administration of barium (Case 2), showing the stomach and duodenum in the abdomen, and loops of small intestine in the right side of the thorax.

Fig. 10. Roentgenograms of the chest and abdomen after administration of barium (Case 2). a, Dorsoventral projection, showing a density with air-filled and barium-filled loops of small intestine in the right side of the thorax, marked displacement of the heart to the left side, lack of visualization of the right side of the diaphragm, and the barium filled colon in the abdomen passing upward into the chest. b, Lateral projection, showing the loops of small intestine and colon posterior to a large, rounded density in the lower half of the right side of the thorax.

no complaints. Physical examinations of the heart and lungs were normal, and the wound was firmly healed. Roentgenograms of the chest revealed progressive lowering of the upward bulge of the right leaf of the diaphragm to the level of the eighth rib posteriorly. At the time of the last examination, the patient's age was 6 years and 7 months, the weight was 42 pounds, and the height was 44½ inches. A roentgenogram of the chest (Fig. 7) showed the right leaf of the diaphragm 2 intercostal spaces higher than the left; the heart and lungs were normal; the opacity of the liver was below the right diaphragm. Fluoroscopic examination revealed diminished respiratory excursion of the right leaf of the diaphragm.

An unusual feature of this case was the late onset of respiratory symptoms. The diagnosis of a congenital right-sided diaphragmatic hernia or eventration was suggested by the physical examination of the chest and abdomen and established by roentgenologic examinations which included barium and pneumoperitoneum studies. The latter procedures outlined the herniated portions of the intestinal tract and liver and a sac-like structure, but differentiation between hernia and eventration of the diaphragm could only be made at operation. Almost the entire diaphragm was replaced by a thin hernial sac. Utilization of the sac in the construction of a diaphragm resulted in a satisfactorily functioning structure.

CASE 2. P. K., a female infant, aged 5 days, was admitted to the Mount Sinai Hospital on August 29, 1950. The infant weighing 7 pounds 1 ounce was born at full term of an uncomplicated pregnancy. Mother, father, and 1 sibling were living and well.

The infant began to breathe spontaneously soon after birth, but cyanosis was noted. Cyanosis continued and required the constant administration of oxygen. The respirations were always rapid and labored. On the first day of life examination of the chest showed dullness to percussion and absent breath sounds on the right side. Bowel sounds were heard in the upper part of the right side of the chest on the second day. There were no changes in the respiratory symptoms and signs during the remaining 3 days. Penicillin was administered.

On the second day of life plain roentgenograms of the chest and abdomen in the dorsoventral and lateral positions (Fig. 8) revealed an opacity filling most of the right side of the thorax within which

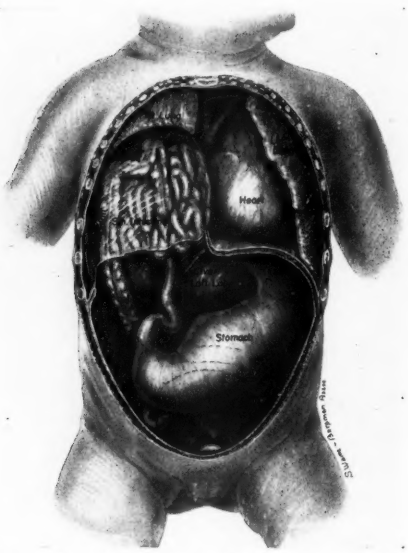


Fig. 11. Drawing illustrating the findings at operation in Case 2.

could be seen areas of translucency suggesting air-filled loops of intestine, the latter passing into the left side of the mediastinum; the heart was displaced to the left side; a large, rounded density in the lower half of the thorax was seen; the stomach was the only gastrointestinal structure visible in the abdomen. On the fourth day roentgenograms of the chest and abdomen, after the administration of barium (Fig. 9), revealed this material in the stomach and duodenum in the abdomen, and in loops of small intestine in the right side of the thorax. The right side of the diaphragm could not be visualized in any of the films.

Physical examination revealed a well nourished infant in severe respiratory distress. Marked cyanosis was noted. The temperature was 99.2 degrees F., the pulse 150, and the respirations 80. She weighed 7 pounds 1 ounce. There were no abnormalities of the head, extremities, abdomen, or genitalia. Examination of the neck showed displacement of the trachea to the left side. Examination of the chest revealed dullness to percussion on the right side below the fifth rib anteriorly and eighth rib posteriorly; above this the percussion note was tympanitic. Breath sounds were absent and bowel sounds were heard in the right side of the thorax. The left border of the heart was at the anterior axillary line.

Examinations of the blood and urine revealed no abnormalities.

Roentgenograms of the chest and abdomen in the dorsoventral and right lateral positions (Fig. 10) revealed most of the findings noted previously, but now the only portion of the intestinal tract visible in the abdomen was the barium-filled colon, which had the form of the sigmoid and descending colon passing upward to the right side of the abdomen where the transverse colon entered the right side of the thorax posterior to the large, rounded density.

Operation was performed on August 29, 1950, a few hours after admission. With the patient under ether anesthesia, a right oblique subcostal incision was made. The only structures visible within the abdomen after opening the peritoneal cavity were the left lobe of the liver, a dilated stomach and first portion of the duodenum, and a small segment of colon (later determined to be the descending colon); these viscera were drawn over to the right of their normal positions. The duodenum and descending colon ran straight up to the region of the right leaf of the diaphragm which was represented by a crescent-shaped shelf along the lateral costal margin. A catheter was passed upward into the thoracic cavity to permit air to enter. The operative findings are illustrated in Figure 11. The small intestine, cecum, ascending colon, and transverse colon were withdrawn from the chest and laid on the abdominal wall covered by warm, moist pads. There was a rudimentary attachment of the mesentery of the small intestine, and the cecum and ascending colon were suspended on a long mesentery. The loops of small intestine and colon were of normal size. The entire right lobe of the liver was then visualized in the right side of the thorax. The herniated portion of the liver was mobilized and brought down into the abdomen. Inspection of the large space in the right side of the chest revealed a thin membrane near the apex of the thorax covering a small, flat, compressed right lung. The hernial sac was incised and withdrawn from the thoracic cavity. This allowed expansion of the lung by positive pressure applied through the anesthesia machine. The sac was attached to a narrow rim of diaphragm along the costal margin laterally, and central tendon of the diaphragm mesially. The defect in the right side of the diaphragm measured about 10 by 7 centimeters in diameter. A portion of the hernial sac was excised, and the free edges approximated and overlapped with superimposed rows of interrupted mattress sutures of silk, extending from the costal margin to the central tendon. The thoracic cavity was emptied of air and bloody fluid by aspiration of the catheter which was withdrawn prior to closure of the last suture. The right lobe of the liver, the small intestine, and the colon were replaced in the peritoneal cavity. The abdomen was closed by sutures of fine steel wire passed in a figure of eight fashion through the peritoneum and oblique and rectus muscles and fascia.

A transfusion of 75 cubic centimeters of citrated blood was administered during operation. The infant was placed in an oxygen cubicle, the indwelling Levin tube was attached to a suction apparatus, and parenteral fluids and penicillin, 300,000 units daily, were started.

During the first postoperative day the highest temperature was 100 degrees F.; the respirations

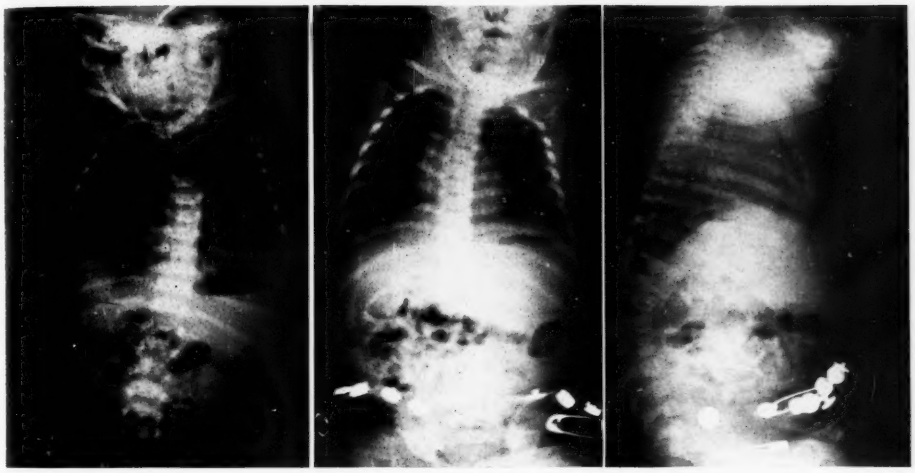


Fig. 12. a Fig. 13. b

Fig. 12. Roentgenogram of the chest and abdomen taken 3 days after the operation (Case 2), showing complete aeration of the lungs, normal position of the diaphragm and heart, and the liver and loops of intestine in the abdomen.

Fig. 13. Roentgenograms of the chest and abdomen taken 2 months after the operation (Case 2). a, Dorsoventral projection, showing normal appearance of the viscera. b, Lateral projection, showing normal position of the diaphragm, and absence of the liver shadow in the thorax seen before operation.

varied from 40 to 50. There was no cyanosis. The abdomen was slightly distended, and there was very little drainage through the gastric tube. Auscultation of the chest revealed normal breath sounds on the right side. The urinary output was adequate. Examination of the blood was normal. During the second postoperative day the highest temperature was 99 degrees F.; the respirations varied from 34 to 40. The infant's condition remained good. Feedings of breast milk were well tolerated, and there were normal bowel movements. On the third postoperative day a roentgenogram of the chest and abdomen (Fig. 12) revealed complete aeration of the lungs, normal position of the diaphragm and heart, and the liver opacity and normal appearing loops of intestine in the abdomen. Gastric siphonage and administration of intravenous fluids and oxygen were discontinued. The infant's uneventful postoperative course continued. The temperature remained normal. Examination of the chest and abdomen revealed no abnormalities. Penicillin was discontinued on the sixth day; a total of 1,650,000 units had been administered. The wound healed by primary union, and the skin sutures were removed on the seventh day. The infant was discharged in excellent condition on the ninth day, weighing 6 pounds 11 ounces.

Follow-up examination 2 months after operation revealed a well nourished and well developed infant. No abnormalities were noted on physical examination. Fluoroscopic examination of the chest revealed normal, symmetrical excursion of both leaves of the diaphragm. Roentgenograms of the chest and abdomen (Fig. 13) showed normal position of both sides of the diaphragm; the heart, lungs, and abdomen were normal.

The clinical manifestations and roentgenologic examination on the second day of life were typical of congenital right-sided diaphragmatic hernia. Administration of barium for diagnosis was unnecessary. The delay in operation for 4 days prior to admission was due to erroneous diagnoses of pulmonary atelectasis and pleural effusion. Fortunately, the infant survived this period. Herniation of the liver was not diagnosed before operation, but was visible in the lateral roentgenograms. The pathology was unusual in that almost the entire diaphragm was replaced by a thin hernial sac which extended to the apex of the chest. Partial excision and repair of the hernial sac resulted in a satisfactorily functioning diaphragm.

CASE 3. D. J., a female infant, aged 48 hours, was admitted to the Mount Sinai Hospital on April 9, 1951. The infant was born at full term of an uncomplicated pregnancy. The mother, father, and 2 siblings were living and well.

The infant cried spontaneously and there were no respiratory symptoms soon after birth. Feedings were

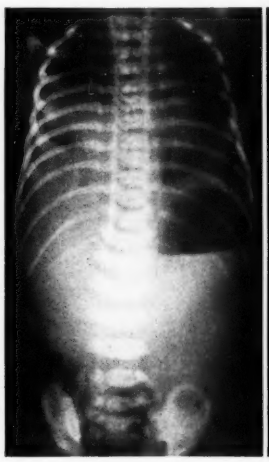


Fig. 14.

a

Fig. 15. b

Fig. 14. Roentgenogram of the chest and abdomen (without the use of a contrast medium, Case 3), showing an opacity with areas of radiolucency filling most of the right side of the thorax, displacement of the heart to the left side, lack of visualization of the right side of the diaphragm, and absence of loops of small intestine in the abdomen.

Fig. 15. Roentgenograms of the chest and abdomen after administration of lipiodol (Case 3). a, Dorsoventral projection, showing the stomach in the abdomen, and loops of small intestine in the right side of the thorax. b, Lateral projection, showing the loops of small intestine posterior to an area of density in the lower half of the right side of the chest.

started after 12 hours, and each feeding was vomited; some of the vomitus was bile-stained. Cyanosis during crying was noted after the onset of vomiting. Normal meconium was passed per rectum. Examination of the chest showed dullness to percussion and absent breath sounds in the lower half of the right side.

A roentgenogram of the chest and abdomen on the first day of life (Fig. 14) revealed a density in the lower right side of the thorax extending to the level of the fifth rib posteriorly within which were areas of radiolucency; the heart and mediastinum were slightly displaced to the left side; the outline of the right side of the diaphragm was not visualized; the stomach, sigmoid colon, and rectum were the only portions of the gastrointestinal tract visible in the abdomen.

Physical examination revealed a well nourished infant, weighing 7 pounds 5 ounces. There was no respiratory distress, but cyanosis was noted during crying. The temperature was 101 degrees F., the pulse 130, and the respirations 50. There were no abnormalities of the head, neck, extremities, or genitalia. Examination of the chest showed dullness to percussion and absent breath sounds on the right side below the scapula; above this the percussion note was tympanitic. The heart was normal. The only abnormality on examination of the abdomen was audible breath sounds.

Examinations of the blood and urine revealed no abnormalities.

Roentgenograms of the chest and abdomen in the dorsoventral and right lateral positions, after a swallow of lipiodol (Fig. 15), showed radio-opaque material in the stomach, in the abdomen, and in loops of small intestine posterior to an area of density in the right side of the thorax.

The infant was placed in an oxygen incubator, intravenous fluids and penicillin were administered, and the stomach was aspirated.

Operation was performed on April 9, 1951, 9 hours after admission. With the patient under cyclopropane anesthesia, a right oblique subcostal incision was made. The only structures visible within the abdomen after opening the peritoneal cavity were part of the right lobe of the liver, the stomach, and a small segment of colon (later determined to be the distal half of the transverse colon). The stomach was drawn to the right side of the abdomen. The duodenum was not visible, seeming to pass behind the right lobe of the liver. The distal half of the transverse colon passed upward along the lateral abdominal wall toward the diaphragm. There was a large defect of the posterior half of the right side of the diaphragm extending from the thoracic wall to the region of the central tendon; the anterior portion of the diaphragm was well developed. A catheter was passed upward into the thoracic cavity to permit air to enter. The operative findings are illustrated in Figure 16.

The small intestine, cecum, ascending colon, and proximal half of the transverse colon were withdrawn

from the chest and laid on the abdominal wall covered by warm, moist pads. There was a rudimentary attachment of the mesentery of the small intestine, and the cecum and ascending colon were suspended on a long mesentery. The colon was of normal size, but the loops of small intestine were empty, small, and collapsed. Further exploration revealed a large section of the right lobe of the liver, including the gallbladder region, situated in the right side of the thorax. These structures were mobilized, withdrawn from the chest, and held outside of the abdomen. The entire defect of the diaphragm could then be visualized; it measured about 4 by 5 centimeters in diameter. There was no hernial sac. The right lung was collapsed, but could be expanded by positive pressure applied through the anesthesia machine. In the absence of a diaphragmatic attachment along the posterior abdominal wall, a broad expanse of the renal fascia covering the anterior surface of the kidney and adrenal gland were dissected free. The defect in the diaphragm was closed with mattress sutures of silk passed through the edges of the renal fascia and diaphragm. The thoracic cavity was emptied of air and bloody fluid by aspiration of the catheter which was withdrawn prior to closure of the last suture. The right lobe of the liver, the small intestine, and the colon were replaced into the peritoneal cavity. The abdomen was closed by sutures of fine steel wire passed in a figure of eight fashion through the peritoneum and oblique and rectus musculature and fascia.

A transfusion of 100 cubic centimeters of citrated blood was administered during and after operation. The infant was placed in an oxygen cubicle, the indwelling Levin tube was attached to a suction apparatus, and parenteral fluids and penicillin continued.

During the first postoperative day the highest temperature was 101 degrees F.; the respirations varied from 36 to 60. There was no respiratory distress or cyanosis. Stool containing oil material was passed. The urinary output was adequate. Examination of the chest revealed slight dullness on percussion and diminished breath sounds in the lower part of the right side. The heart was in its normal position. During the second postoperative day the highest temperature was 100.2 degrees F. The infant's condition remained good. Feedings were well tolerated, and there were normal bowel movements. Examination of the chest showed no change from the previous examination. Gastric siphonage and administration of intravenous fluids and oxygen were discontinued. Roentgenograms of the chest and abdomen in the dorsoventral and right lateral positions, 26 hours after operation (Fig. 17), revealed good aeration of the lungs, normal position of the diaphragm and heart, absence of the density in the right side of the thorax, and loops of intestine in the abdomen. The infant's uneventful postoperative course continued. The temperature was normal except for a slight rise on the fourth day. Examination of the chest and abdomen revealed no abnormalities. Fluoroscopic examination of the chest on the fourth day revealed normal, symmetrical excursion of both leaves of the diaphragm. A roentgenogram of the chest and abdomen showed no abnormalities. The wound healed by primary union, and the skin sutures were removed on the fifth day. Penicillin was discontinued on the sixth day; a total of 1.5 million units had been administered. The infant was discharged in excellent condition on the eighth postoperative day.

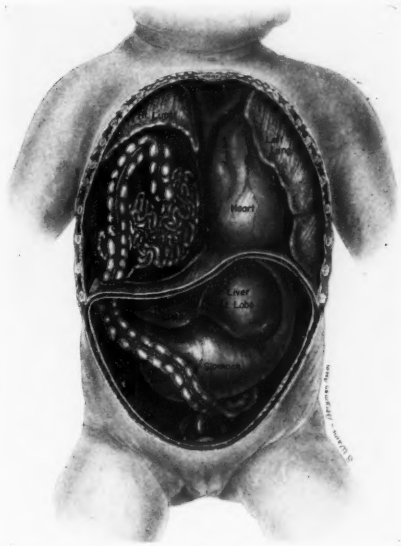
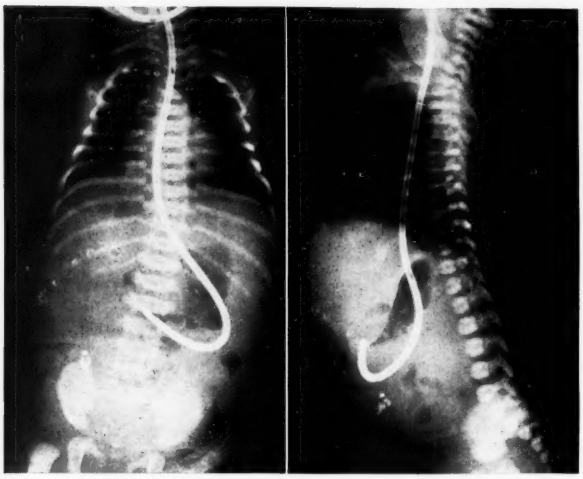


Fig. 16. Drawing illustrating the findings at operation in Case 3.

Follow-up examination, 5 months after operation, revealed a well nourished and well developed infant. The weight was 16 pounds 6 ounces. No abnormalities were noted on physical examination. Fluoroscopic examination of the chest revealed normal, symmetrical excursion of the diaphragm. A roentgenogram of the chest and abdomen (Fig. 18) showed normal position of both sides of the diaphragm; the heart, lungs, and abdomen appeared normal. Roentgenograms of the abdomen, after the administration of barium (Fig. 19), showed the stomach and loops of small intestine filled with the opaque material; the density of the liver was visualized; the contours of the diaphragm were well outlined in the lateral view.

The history was unusual for this age group in that the first symptom was vomiting. The roentgenogram taken prior to admission to the hospital was quite characteristic of con-



a b

Fig. 17. Roentgenograms of the chest and abdomen taken 26 hours after the operation (Case 3). a, Dorsoventral projection, showing complete aeration of the lungs, normal position of the diaphragm and heart, and the liver and loops of intestine in the abdomen. b, Lateral projection, showing absence of the liver shadow in the thorax seen before operation.

genital right-sided diaphragmatic hernia, but loops of intestine were not seen in the first roentgenograms after admission; these were visualized after a swallow of radio-opaque material. Herniation of the liver was visible in a lateral roentgenogram. Operation was performed early in the neonatal period. There was no hernial sac. Utilization of the renal fascia in the repair of the large defect in the posterior half of the diaphragm resulted in a satisfactorily functioning diaphragm.

DISCUSSION

The surgical management of congenital right-sided diaphragmatic hernia has rarely been the subject of published reports (26 cases in the literature). Experience with 3 cases prompted a review of the literature, and 9 other cases were found in which the liver and intestines were the structures involved in the hernia (Table I). An analysis of these cases will be presented.

The age of the patients at operation varied from 6 hours to 4 years as follows: 6 hours to 5 days in 4, 13 to 16 days in 2, 2 to 10 months in 3, and 4 years in 3.

There were 5 females, 4 males, and in 3 the sex was not mentioned.

Analysis of the symptoms of the 4 infants treated in the first week of life showed cyanosis in all patients, combined with respiratory difficulties—rapid respirations and dyspnea—in 3. Vomiting was noted in 2 cases, 12 hours and 4 days after birth. Difficulty with nursing was present in 1 case. Examination of these infants revealed dullness on percussion of the right side of the chest in 3 patients. Absence of breath sounds on the right side and cardiac displacement to the left side were found in 2 infants. Bowel sounds were heard in the right side of the chest in 1 patient. One infant was in a state of shock.

Cyanosis was a symptom in both infants, 13 and 16 days of age, combined with shallow, rapid respirations and vomiting in 1, and difficulty in nursing and poor weight gain in the other. The infant with respiratory symptoms was in a state of shock, with absent breath sounds and audible bowel sounds in the right side of the thorax, and cardiac displacement to the left side. Physical signs were not mentioned in the other patient.

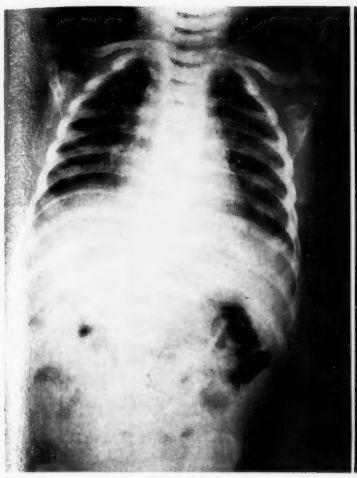
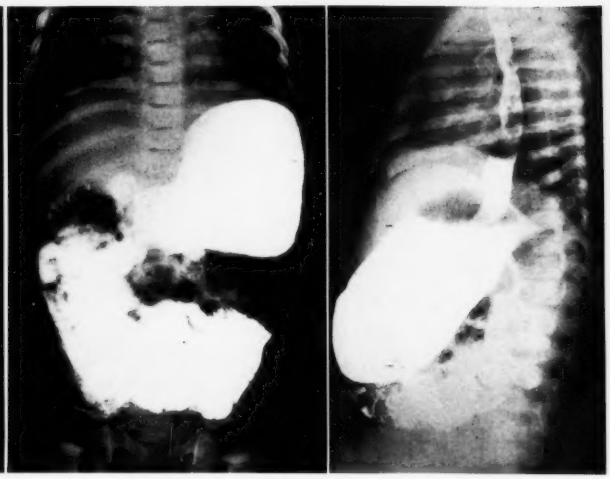


Fig. 18.

Fig. 18. Roentgenogram of the chest and abdomen (without the use of a contrast medium) taken 5 months after the operation (Case 3), showing normal appearance of the viscera and diaphragm.



a Fig. 19. b

Fig. 19. Roentgenograms of the chest and abdomen (after administration of barium) taken 5 months after the operation (Case 3). a, Dorsoventral projection, showing the barium filled stomach and small intestine and the liver in the abdomen. b, Lateral projection, showing normal position and contour of both leaves of the diaphragm.

Dyspnea and cyanosis were the presenting symptoms in the 3 infants, 2 to 10 months of age, in 2 dating from birth and noted for only 1 week in the 10 month old patient. Poor weight gain and intermittent fever were noted in 2 infants, and vomiting in 1. Dullness or tympany on percussion of the right side of the chest were elicited in all these infants. Bowel sounds were heard in the right side of the chest in 2 patients, and absent breath sounds on the right side and cardiac displacement to the left were present in 1 patient.

The symptoms of the 3 children, aged 4 years, differed in each—vomiting and poor weight gain since birth, cough and fever for 1 year, and dyspnea and cyanosis. Diminished or absent breath sounds in the right side of the chest were noted in 2 patients, and bowel sounds on this side were heard in 2 patients. Dullness to percussion on the right side and cardiac displacement to the left were elicited in 1 child. Physical signs were not mentioned in 1 patient.

Various errors in diagnosis were made prior to admission to the hospital: atelectasis in an infant 5 days old, pulmonary tuberculosis and pneumonia in 2 of the 4 year old children. The diagnosis of right-sided diaphragmatic hernia was made by roentgenologic examination in 10 cases; roentgenograms were not mentioned in 2 cases reported in tabular form. Plain films alone were made in 4 cases, and combined with studies with opaque media in 6. The roentgenologic findings took the form of air or media-filled loops of intestine in the right side of the thorax. Displacement of the heart to the left side was commonly noted. The upward extension of the loops of intestine was limited in 3 of the 4 cases in which a sac was present. Pneumoperitoneum outlined the sac in 1 case of the author, but a differential diagnosis between hernia and eventration of the diaphragm was not made. The roentgenologic diagnosis of herniation of the liver into the thorax was made in this case. It is surprising that involvement of the liver was not diagnosed in other x-ray reports. The density of this organ in the right side of the thorax is, in itself, not characteristic, but when combined with loops of bowel in this region is of diagnostic value.

The amount of liver found at operation in the thoracic cavity was described as follows:

TABLE I.—PATIENTS OPERATED UPON FOR CONGENITAL RIGHT SIDED DIAPHRAGMATIC HERNIA OF LIVER AND INTESTINES

Author	Age, sex	Clinical manifestations*	X-ray	Herniated viscera	Site of defect	Sac	Operation	Anesthesia	Result
Truesdale (1939)	4 yr. F	Vomiting and poor weight gain since birth; bowel sounds, absent breath sounds, and cardiac displacement	Positive (plain and barium)	Small intestine, cecum, appendix, ascending colon, ½ transverse colon, and left lobe of liver	Large anterior	+	Abdominal approach (2 stages); diaphragm repair, plication of sac, and attachment to thoracic wall		Cure
Ladd and Gross (1940)	13 days M	Cyanosis and shallow, rapid respirations since birth, and vomiting for 2 days; shock, bowel sounds, absent breath sounds, and cardiac displacement	Positive (plain)	Distal half of stomach, duodenum, small intestine, cecum, appendix, ascending colon, ½ transverse colon, and right lobe of liver	Large posterior	o	Abdominal approach; cervical phrenic crush, diaphragm repair, and abdominal wall repair 4 mos. later	Cyclopropane	Cure
Wyatt (1941)	6 hr. M	Cyanosis and shallow rapid respirations since birth; shock	Positive (plain)	Almost entire gastrointestinal tract and ½ liver	Large posterior	o	Abdominal approach; diaphragm repair	Oxygen	Death (termination of operation)
McDowell and Tondra (1946)	5 days M	Cyanosis and poor nursing since birth, and vomiting and dyspnea for 1 day; percussion dullness and cardiac displacement	Positive (plain)	Part of small intestine, most of colon, and ¼ right lobe of liver	Large anteromesial	o	Thoracic approach; diaphragm repair	Local	Death (termination of operation)
Belsey and Apley (1949)	4 yr. F	Cough and fever for 1 yr.; poor movement, percussion dullness, diminished breath sounds, and bowel sounds	Positive (plain and barium)	Part of duodenum, small intestine, cecum, appendix, ascending colon, ½ transverse colon, and liver†	Large posterior	o	Thoracic approach; diaphragm detachment, repair, and reattachment to thoracic wall		Cure
Zeller (1950)	4 yr.	Dyspnea and cyanosis		Most of small intestine and ½ liver	Large posterior	o	Abdominal approach; diaphragm repair		Cure
Zeller (1950)	16 days	Cyanosis, difficult nursing, and poor weight gain		Part of small intestine and major part of liver	Large posterior	+	Abdominal approach; diaphragm repair, and abdominal wall repair 9 days later		Cure
Baumgartner and Scott (1950)	2 mos.	Dyspnea, cyanosis, and vomiting; percussion dullness	Positive (plain)	Small intestine, cecum, appendix, ascending colon, and right lobe of liver	Foramen of Bochdalek	o	Abdominal approach; diaphragm repair		Cure
Howard (1950)	6 mos. M	Dyspnea, cyanosis, and poor weight gain since birth, and fever for 2 mos.; percussion tympany, bowel and cardiac displacement	Positive (plain and barium)	Small intestine, cecum, appendix, ascending colon, transverse colon, part of descending colon, and part of right lobe of liver	Foramen of Bochdalek	o	Abdominal approach; diaphragm repair	Cyclopropane and ether	Cure
Arnheim (1952)	10 mos. F	Poor weight gain since birth, and fever, cyanosis, and dyspnea for 1 wk.; poor expansion, percussion dullness and tympany, absent breath sounds, and bowel sounds	Positive (plain, barium, and pneumoperitoneum)	Cecum, appendix, ascending colon, hepatic flexure of colon, and right lobe of liver	Almost complete absence of hemidiaphragm	+	Abdominal approach; plication of sac and attachment to edges of diaphragm	Ether	Cure
Arnheim (1952)	5 days F	Cyanosis and rapid labored respirations since birth; percussion dullness and tympany, absent breath sounds, bowel sounds, and cardiac displacement	Positive (plain and barium)	Small intestine, cecum, appendix, ascending colon, transverse colon and right lobe of liver	Almost complete absence of hemidiaphragm	+	Abdominal approach; partial excision and plication of sac	Ether	Cure
Arnheim (1952)	2 days F	Vomiting and cyanosis for 36 hr.; percussion dullness and tympany, and absent breath sounds	Positive (plain and lipiodol)	Small intestine, cecum, appendix, ascending colon, ½ transverse colon, and part of right lobe of liver	Large posterior	o	Abdominal approach; diaphragm repair utilizing renal fascia	Cyclopropane	Cure

*Pulmonary signs on right side and cardiac displacement to left side.

†Amount of liver not stated.

right lobe in 4 patients, part of right lobe in 3, one-half in 2, major part in 1, left lobe in 1, and not stated in 1.

The small intestine was herniated in 11 cases, in association with a rudimentary attachment of its mesentery. The cecum, appendix, ascending colon, and part of the transverse colon were involved in the hernia in 10 cases, in association with incomplete fusion of the mesentery of the right portion of the colon. Parts of the descending colon and duodenum were noted in 2 cases, and a portion of the stomach in 2.

The sites of the defects in the right diaphragm were described as large posterior in 6 cases, foramen of Bochdalek in 2, almost complete absence of hemidiaphragm in 2, and large anterior in 2. A hernial sac was absent in 8 cases, and present in 4.

The operative approach was abdominal in 10 patients and thoracic in 2. A variety of repairs of the diaphragm was carried out. The usual type of closure was performed in 7 cases, preceded by phrenic crush in 1, and followed by secondary closure of abdominal wall in 2.

In 2 cases of the author and in 1 other case in which a sac was present, a diaphragm was constructed by plication of the sac, as previously described. In 1 case the diaphragm was detached from the costal margin, the defect repaired, and the diaphragm reattached to the thoracic wall at a higher level. The renal fascia was used for repair in 1 of the author's cases.

A closed system of anesthesia (cyclopropane or ether) was used in 5 patients. The type of anesthesia was not noted in 5 cases. The 6 hour old infant was in such poor condition that only oxygen was administered. Local anesthesia was used in 1 patient.

Two patients died at the termination of operation (operative mortality 16.6 per cent). These infants were 6 hours and 5 days of age. The first patient was in very poor condition before operation. Operation in the second patient was performed under local anesthesia by the thoracic approach.

SUMMARY

The surgical pathology and treatment of the most common types of congenital diaphragmatic hernia in infancy and childhood were reviewed.

An analysis of reported cases of congenital right-sided diaphragmatic hernia of the liver and intestines treated surgically is presented.

A technique for the plastic repair of almost complete absence of a hemidiaphragm utilizing the hernial sac is described.

Three cases of congenital right-sided diaphragmatic hernia of the liver and intestines successfully treated surgically are reported.

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THE REPAIR OF DIAPHRAGMATIC DEFECTS WITH CUTIS GRAFTS

An Experimental Study

ERNEST D. GEEVER, M.D., Whipple, Arizona, and
K. ALVIN MERENDINO, M.D., F.A.C.S., Seattle, Washington

THE closure of most diaphragmatic defects is simply a matter of approximating free edges. However, in large congenital defects, eventration, diaphragmatic resection for primary malignancy of the diaphragm, or secondary to pulmonary or abdominal cancers, closure may be impossible without alternative procedures. Such alternatives have involved mobilization of the renal fascia, interposition of divided latissimus dorsi muscle, or partial resection of the lower ribs in order to reduce the defect size.

It is apparent that the interposition of a suitable grafting tissue would assist greatly in the closure of large defects. A satisfactory diaphragmatic substitute should be readily available, in quantity, of good tensile strength, pliable, and autogenous. Elasticity is desirable but not necessary. These characteristics appear to be inherent in cutis tissue.

In this country, Cannaday has been one of the strongest advocates of the use of cutis in the repair of large external hernia. In most instances, cutis tissue has been used as a supporting patch over the suture line of an otherwise adequate fascial repair and seldom as a means of bridging a gap. Harkins, incidental to a larger study, reported the use of small (2.5 by 4 cm. and 4 by 6 cm.) free cutis grafts in the repair of experimental diaphragmatic defects in 2 dogs surviving 2.5 and 4 months. Both healed without incident. Recently, Metheny utilized a free cutis graft in the repair of a diaphragmatic defect secondary to the removal of a gastric cancer without complication.

This latter experience stimulated us to evaluate experimentally the behavior of cutis buried in the body cavity as a diaphragmatic substitute.

From the Department of Surgery, University of Washington School of Medicine, Seattle, Washington. Supported by State of Washington Initiative 171 Funds for research in biology and medicine.

GENERAL METHOD OF STUDY

Sixty-four defects of varying size were manufactured and immediately repaired in 32 dogs. Half the number of defects were repaired with free cutis autografts and the other half were repaired with pedicle cutis autografts. The defects averaged 75 square centimeters (52 operations). In addition, total (bilateral) diaphragmatic resection[1] was performed on 6 animals, 3 being repaired with free grafts and 3 with pedicle grafts. These defects averaged 180 square centimeters (12 operations).

The smaller defects were made in continuity with the chest wall to simulate congenital occurrences. No special attempt was made to reduce the bacterial flora of the donor sites. Only routine antiseptics and ether were used. The animals were periodically fluoroscoped postoperatively. Necropsy specimens of the graft and neighboring viscera were taken from the fourth postoperative day and at intervals up to 4 months. India ink injections were made in some animals in order to determine the source of blood supply to the graft.

SPECIFIC PROCEDURE

The free graft. After nembutal anesthesia, the animal was clipped and shaved closely. An endotracheal tube was inserted and connected to a Palmer respirator. The skin was cut by means of a Padgett dermatome using the split-split method. The initial depth was 0.030 inch. After adjusting the blade, the dermatome was run back at 0.010 inch. This maneuver left 2 skin flaps: one, the epidermis

[1]Measurements of completely extirpated diaphragms in comparable sized dogs indicated a 90 per cent removal of the total diaphragm after the bilateral procedure.

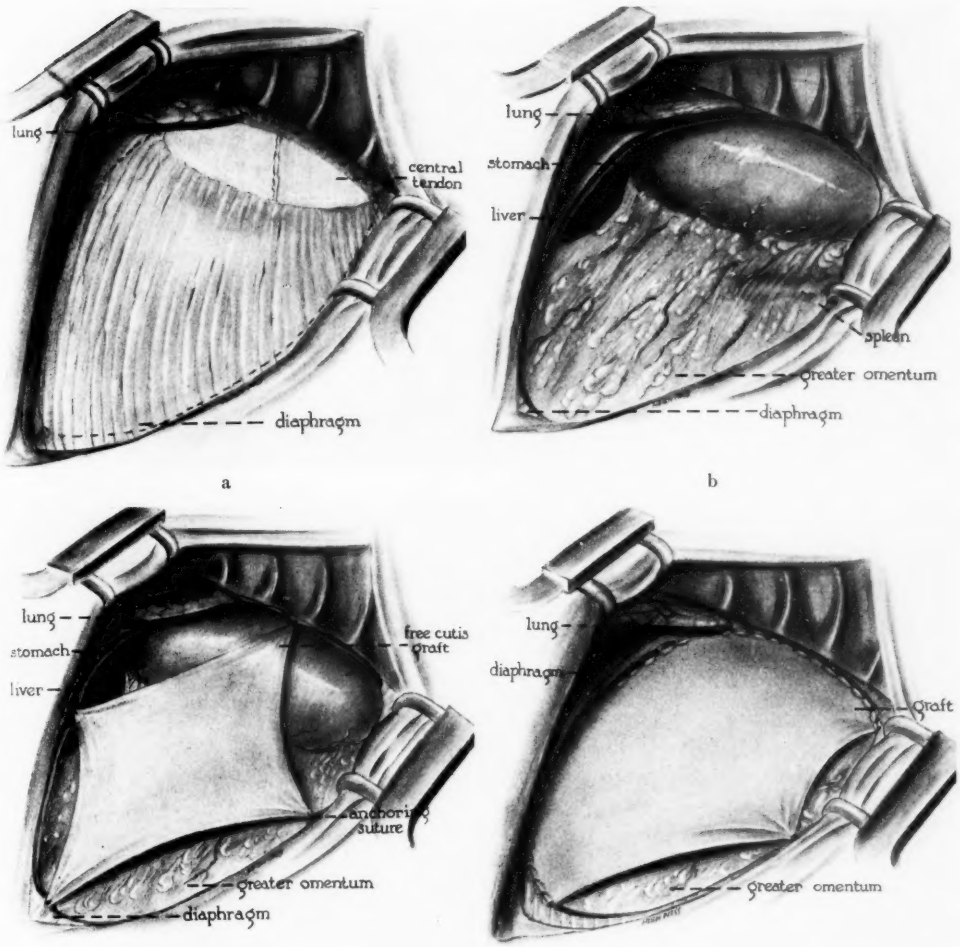

Fig. 1. Free cutis graft. a, Exposure of the left diaphragm through the tenth intercostal space. The dotted lines enclose the area of the diaphragm to be excised. b, The diaphragmatic defect has been created, exposing the abdominal viscera. c, Insertion of the cutis graft, which has been fixed by 4 interrupted silk sutures at opposing ends. d, Fixation of the cutis graft to diaphragmatic edges is being completed by running over and over sutures.

of 0.010 inch thickness and the other, the cutis of 0.020 inch thickness. The cutis was detached and kept in saline. The chest then was opened through the ninth or tenth interspace, and the defect created. The graft was anchored at opposite portions, in order to produce equivalent stretch, and the defect was closed with running sutures (Fig. 1, a, b, c, d). Measurements across two diameters of the repaired defects were taken before chest closure. Crystalline penicillin, 200,000 units, was instilled into the pleural cavity and into the wound. The wound was closed around a suction tip after the lung was inflated. The epidermal flap then was replaced over the donor site, and sutured in place. Postopera-

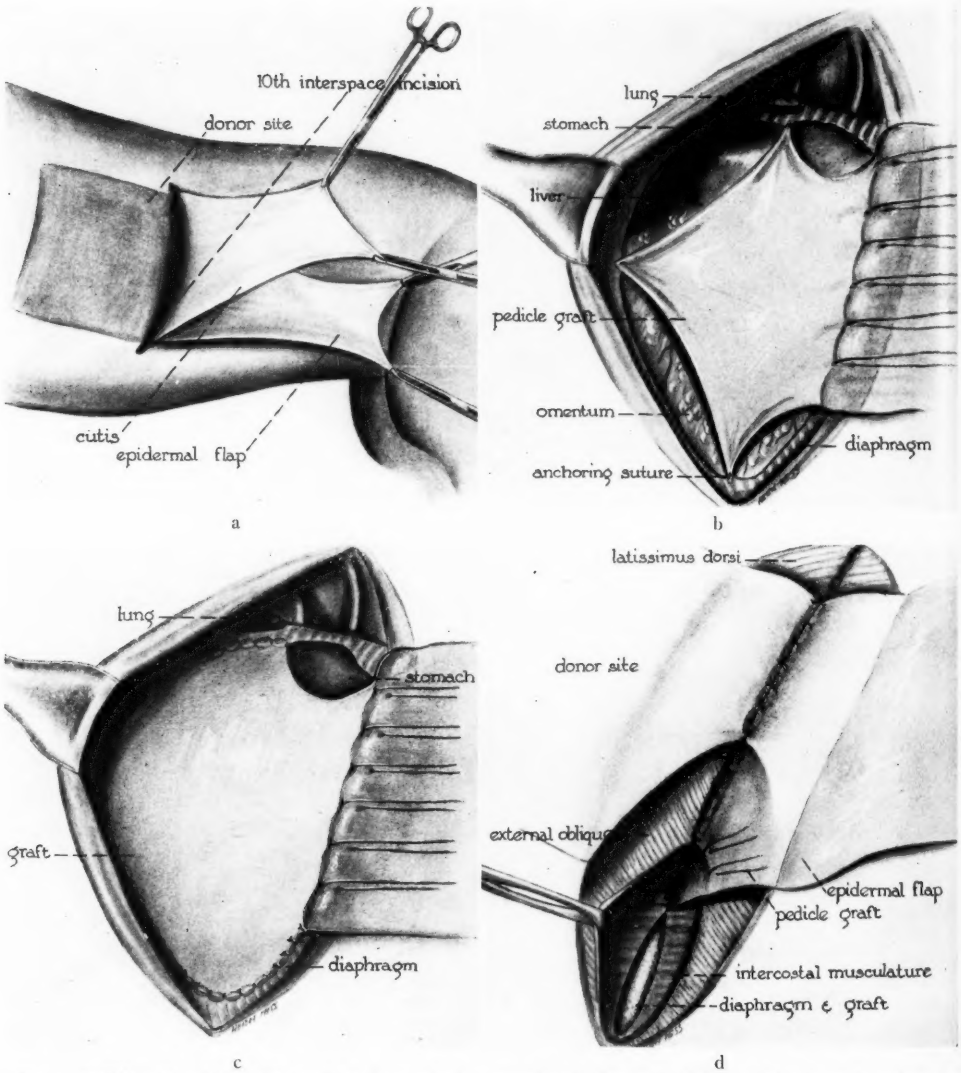

Fig. 2. Pedicle cutis graft. a, The graft has been raised, resplit, and the thoracotomy incision made. b, The diaphragmatic defect has been made and the cutis is being sutured into position. c, The repair of the diaphragm almost completed. One end of the untied sutures is rethreaded and the opposing musculature of the thoracic wall is sutured to the base of the cutis in order to effect the chest closure. d, A further stage in the closure of the incision.

tively, the animals received daily penicillin in beeswax, 300,000 units, for a period of 3 days.

The pedicle graft. After a satisfactory initial flap was raised, the dermatome was reset and a cutis flap cut as described previously in the free graft. The chest incision then was made through the donor site close to the attached base of the cutis flap, and the defect was developed. Before the cutis flap was turned down into the wound, several interrupted

sutures were placed through the graft catching the underlying musculature of the chest wall as the latter was not available again under observation. Care was taken to interrupt only a minimum of blood flow which might be carried through the pedicle. The base of the cutis flap was sutured first. The free ends were anchored at opposing points. The remainder of the closure was effected by running sutures. The wound closure proceeded from either angle in a routine fashion until the graft was approached, whereupon the previously inserted base sutures were rethreaded and passed through corresponding muscular layers in the exposed side of the wound (Fig. 2, a, b, c, d). Chest aspiration and penicillin instillation, as described previously, were carried out before complete closure. Similarly, these animals received daily 300,000 units of penicillin in beeswax through the third postoperative day.

The technique of the pedicle repair posed several problems. The first was related to the confinement of the donor site adjacent to the point of entry into the chest. This placement of the donor site presented the problem of raising the initial skin flap over the complex curvatures of the thoracic cage. This necessity frequently resulted in a failure to cut the skin uniformly.

The pedicle graft preparation was utilized in the hope that perhaps a modicum of blood might flow through the base of the pedicle. As a consequence, one might expect fewer failures and less shrinkage of the cutis when compared to results obtained with the free cutis grafts.

In the reconstruction of the diaphragm, it was considered advisable to deviate from Swenson's suggestion that better results may be obtained with the cutis under tension. Because of the diaphragmatic curvature, tension was obtained only at the expense of this curvature. Consequently, this maneuver resulted in the absolute immobilization and flattening of the diaphragmatic leaf. Therefore, the desire to produce tension in the partially resected animals was foregone; however, the grafts were inserted under considerable tension in the total (bilateral) diaphragmatic resections.

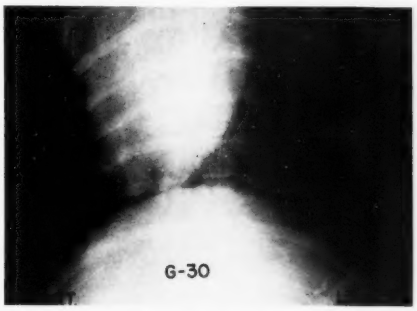

Fig. 3. Dog No. G-30. Left hemiresection replaced by a free cutis graft. The graft measured 14 by 8 centimeters after being sutured into the defect. No surgery was performed on the right side. The roentgenogram was taken on the fourteenth postoperative day.

RESULTS

Roentgenography. The function of the grafted diaphragm was studied by fluoroscopy and roentgenography before and after each procedure. The mobility was studied by means of an interposed grid.[1] While under the fluoroscope, the animals were hyperventilated by rebreathing into a snout bag. Electrical stimulation of the phrenic nerve was attempted but discarded as being unsatisfactory for this purpose. In all cases there was a distinct

[1]Grid squares measured +1 centimeter.

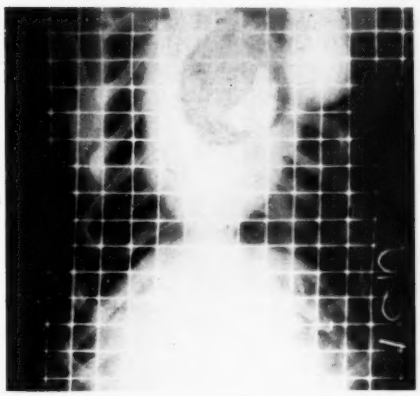

Fig. 4. Dog No. G-37. "Total" left diaphragmatic resection repaired by a pedicle cutis graft (in place measured 18 by 9 cm.) May 17, 1951. On May 24, 1951, a right "total" diaphragmatic resection was done and a pedicled cutis graft (19 by 9 cm.) was utilized in the repair. The roentgenogram was taken 3 weeks after the first operation and 2 weeks after the second.

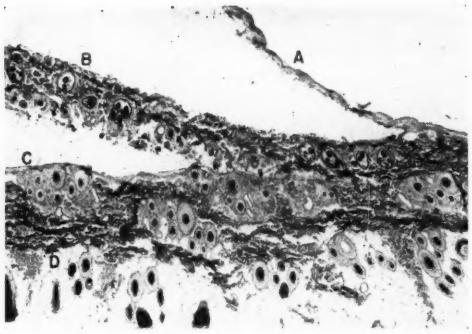

Fig. 5. Photomicrograph of normal dog skin. *A*, The epidermis utilized for skin graft; 0.010 inch. *B*, The cutis used for the repair of the diaphragm; 0.020 inch. *C*, The remaining cutis of the donor bed; approximately 0.030 inch. *D*, Subcutaneous tissue, × 19.

diminution of motility of the grafted leaf. The decrease in motion was in direct proportion to the size of the defect. No evidence of diaphragmatic motion was observed in total (bilateral) resected animals. There was, however, no instance of paradoxical motion. The diaphragmatic curvature was indistinct only in those animals with marked pleural reaction. The diaphragmatic curvature was reduplicated accurately even in total (bilateral) resected animals (Figs. 3 and 4).

Observations of physical capability. In the absence of infection, the animals were normally active within 1 to 2 weeks' time. "Totally" resected animals required the longest period to recover. However, after convalescence, these latter animals exhibited normal healthy activity. Air hunger was not apparent.

Autopsy findings. Groups of 10 animals (5 free grafts and 5 pedicle grafts) were sacrificed on the fourth, ninth, fourteenth, and thirtieth postoperative days. Smaller groups of animals were sacrificed 2, 3, and 4 months after surgery. Four animals have been allowed to survive for a long term study.

TABLE I.—AVERAGE INCIDENCE OF INFECTION*

	Per cent over-all incidence of infection	Per cent wound infection and dehiscence	Per cent empyema
Entire series.....	14	10.9	6.2†
Pedicle graft group...	25	15.6	9.4
Free graft group.....	3	0	3.0

*Superficial infection from factors other than the graft not included.
†Wound infection and dehiscence in conjunction with empyema explains discrepancies in totalling percentages.

Graft.—Of 64 procedures, there were 2 instances of partial failure and partial necrosis of the graft; one graft failed for 40 per cent of its circumference, the other for 10 per cent. The larger separation was associated with empyema, the smaller with a fistula connected to a localized abscess over the graft (empyema necessitans). True hernia did not develop in the larger separation because of the inflammatory agglutination of the lung to this area and the inactivity of the animal from the day of surgery until the ninth postoperative day when it expired. The 10 per cent separation resulted in a true hernia involving the entire stomach which had become obstructed in the chest. This animal expired 72 days after surgery. Both failures occurred with the pedicle graft.

The over-all incidence of infection for the entire series was 14 per cent. In the pedicle graft series, infection was a serious and practically unavoidable complication. The major difficulty was the fact that the epidermis, cutis, and donor bed were in continuity with the surface of the animal. Therefore, daily contamination of the operation area occurred. The only instances of partial failure and necrosis (2 cases) occurred in the pedicle graft group, both associated with infection. It is assumed that the presence of infection initiated the failure and necrosis. In contrast with this experience, the incidence of infection in the free graft series was 1 in 30 operations (3 per cent) (Table I).

Experiences with reversal of the graft surface; i.e., epidermal surface toward the pleural cavity versus epidermal surface toward the peritoneal cavity, were inconclusive. Such an experience would be expected, since it is apparent from the microscopy of the cutis flap, that the cross sectional area of exposed epithelial elements is about equal on either side (See "Histopathology").

The graft shrinkage was computed by the comparison of the two diameter measurements taken at surgery with those taken at postmortem. Shrinkage in 1 month specimens was found to be 12.6 per cent; in 2 month specimens, 28.3 per cent; in 3 month specimens,

31.6 per cent; in 4 month specimens, 26.8 per cent.

From these data the maximum shrinkage is approximately 30 per cent. Furthermore, this shrinkage appears to be complete soon after the second month. However, upon microscopic study at this period, fibroblasts persist within the graft. Their subsequent maturation is by no means complete in 4 months. Additional shrinkage probably occurs at the completion of this process. Specimens from 6 months and 1 year old animals should help clarify this point.

Adhesions.—The graft was adherent to adjacent structures in every instance although in varying degree. On the peritoneal side of the graft, the liver, fundus of the stomach, spleen, and omentum were involved singly or collectively. On the pleural side, the graft was usually adherent to chest wall, diaphragmatic surface of lung, or pericardium.

Adhesions to the peritoneal viscera produced no apparent physiologic detriment whereas pulmonary adherence possibly caused some functional limitation. However, the vascularity of the adhesions was highly instrumental in allowing the consistent survival of the grafts. The division of organized adhesions was followed by brisk oozing from the pleural or peritoneal sides of the graft. Certain intercostal arteries, the internal mammary and the phrenic arteries; as well as the left gastric, left gastroepiploic arteries, and smaller branches of the omental arteries contributing to the circulation of the graft were injected individually in different animals with India ink at various postoperative periods. The animal was allowed to live for 30 minutes after injection before necropsy specimens of the graft were obtained. The results from the injection of vessels other than the intercostal and omental vessels were inconclusive, probably because of their distance from the graft which resulted in excessive dilution of the ink. In addition, India ink was injected close to the dermal attachment of the pedicle graft in several animals. No definite vessels were apparent here; the ink was injected intracutaneously. Migration of the ink beyond 5 millimeters from the point of injection was not demonstrated microscopically.

It would appear that the vascularity and hence the survival of the graft is provided by the blood vessels supplying the diaphragm and supplemented by vessels traversing organized adhesions. No credit for the viability of the graft can be attributed to the pedicle type procedure.

In general, the cutis causes only an inflammatory reaction within the pleural cavity. It is the rule to find little more evidence of inflammation within the pleural cavities than that expected following simple thoracotomy. The explanation for this fact appears to be the rapidity with which pleural exudate covers the graft. By the fourth postoperative day the graft is completely covered with a gelatinous membrane. The inference is not intended that the gelatinous (serofibrinous) coating represents pleuralization nor that it, in itself, prevents adherence of the lung to the graft. It is possible that it serves to contain bacteria within the follicles of the graft.

HISTOPATHOLOGY

Graft. Figure 5 localizes within the integument the depths of the tissues utilized experimentally. It was noted that 0.010 inch adequately removed the epidermis, and that 0.020 inch of cutis represented about one-half of the available derma. Therefore, after an initial 0.030 inch cut, there remained an additional 0.030 inch (±) of cutis. The remaining donor bed was observed to contain an undisturbed bulk of the hair follicles which permitted these donor sites to regrow a sparse amount of hair. In addition, these follicles provided foci from which epithelialization occurred in the event of an epidermal flap slough. These follicles were relied upon to heal many of the donor sites; the epidermal flap being lost frequently because of the animals' oral ministrations to the area, and the inability to prevent contamination postoperatively.

Early, the hair follicles underwent enlargement with cystic degeneration. These cysts were lined with corneum. Atrophy and hyalination occurred by the fourth month. These cysts were not epidermal islands left behind. They exemplified the ability of the follicle epithelium to reduplicate epidermal layers including the stratum corneum. Granulomatous

aggregations (without necrotic centers) in juxtaposition to the hair follicles dot the graft and tend to persist through the fourth month specimens. These microscopic areas consist mainly of lymphocytes, monocytes, fibroblasts, and rare giant cells of the foreign body type. The granulomas increased in number as the hair follicles reduced in number. These granulomatous areas probably represent an intermediate stage of the disintegration process of the follicle itself. Identifiable hair roots were absent in 4 month specimens.

It has been observed that fibrocytes retain the ability to revert back to the immature fibroblast under stimulation (1). It is probable that the fibrosis of the graft occurs from multiple foci of converted fibroblasts. These foci coalesce and together with the infiltrating fibroblasts from the periphery of the diaphragm complete the fibrosis. Islands of fibroblasts consistently are evident in microscopic sections and a distinct increase in the fibrous tissue elements is present. At the periphery of the graft, fibroblasts in sheets and whorls infiltrate the granulation tissue at the junction of the cutis and diaphragm. However, these tissue elements appear to remain localized to this area as late as 4 months.

Small "abscesses" identified by polymorphonuclear accumulations around necrotic centers were seen occasionally in the early specimens; i.e., 4 days, 9 days, and 14 days postoperatively. Their source was probably the sebaceous gland associated with the hair follicle. The resolution of these abscesses was proceeding rapidly by 1 month although some smoldering foci are still present.

Adhesions. The organized adhesions contained numerous capillaries crossing between the graft and the omentum, serosa of the upper stomach, capsule of liver, and spleen. On the pleural side, the serofibrinous exudate was infiltrated by a syncytium of endothelial and epithelioid cells. Overlying this exudative layer was a less vascular strata of mature fibrous tissue constituting pleuralization.

Viscera. The most noteworthy changes which occurred in adjacent viscera appeared in the lung. In addition to the pleural thickening, there were seen occasionally minor areas of atelectasis and pneumonitis. This increase of cellular elements in the lung parenchyma consisted of chronic inflammatory cells. Rarely, the cellular elements extended over into the alveolar spaces and presented a picture not unlike "alveolar" pneumonia. All the changes noted occurred in the lower lobe immediately adjacent to the diaphragmatic surface.

No abnormal microscopic changes were noted in the architecture of the adhered liver, spleen, or stomach.

POSSIBLE APPLICATIONS IN MAN

In most congenital diaphragmatic defects, the repair merely consists of reapposition of the edges of the defect. However, in hemiagenesis of the diaphragm or variations thereof wherein accessory tissue must be utilized, the free cutis graft is a readily available substitute in quantity. Likewise, in certain instances of eventration, the cutis graft should be valuable. Following the resection of primary diaphragmatic tumors, or in abdominal and pulmonary carcinomas of the lower lung with secondary invasion of the diaphragm, the use of a cutis substitute should be satisfactory.

In situations wherein pneumonectomy and pleurectomy may be indicated for pulmonary carcinoma, the inability to remove cleanly the pleura from the diaphragm has defeated the purpose of this radical approach. In such instances the sacrifice of the diaphragm with cutis substitution offers an alternative procedure of merit. However, when a total pneumonectomy is performed, one should anticipate the increased possibility of an empyema and the necessity for a thoracoplasty.

In lobectomy cases, an additional safety factor would appear to be present. The remaining lobes, upon expansion, tend to buttress the grafts and supply a potential source of blood flow to assist in the preservation and integrity of the graft. In addition, the pulmonic tissue and visceral pleura would appear to contain any potential contamination from the graft.

In considering the application of cutis grafts to humans, the choice of the donor site may be conditioned by the thickness of the cutis desired. Certainly, the skin of the back

should be sufficiently thick to obtain a cutis graft to withstand any stress or strain demanded of soft tissue. In children, it might be necessary to utilize the full thickness of the cutis with the exception of the epidermal layer.

SUMMARY AND DISCUSSION

Cutis grafts have been shown to provide a satisfactory barrier between the pleural and peritoneal cavities. The free cutis grafts have proved in the experimental animal to be superior to the pedicle cutis graft.

The possible advantage that some blood flow might be carried through the pedicle base proved to be unfounded. Furthermore, with the pedicle cutis graft, the rate of infection was consistently higher. This observation is related to the fact that the base of the pedicle graft reaches to the skin. In dogs, contamination cannot be prevented. A major disadvantage of the pedicle cutis graft is related to the necessity of knowing the size of the defect prior to the placement of the skin incision. However, the free cutis graft has proved to be more efficacious than the pedicle cutis in providing a barrier and has the following advantages: no limitation exists regarding the amount of the donor cutis, the size of the graft may be obtained according to the pattern of the defect under direct vision, and the incidence of important infection is practically nonexistent.

Graft shrinkage of approximately 30 per cent can be anticipated by the fourth month postoperatively. Nevertheless, the contour of the reconstructed diaphragm remained satisfactory. In fact, it was impossible in most instances to determine by x-ray the recipient area. Further minimal shrinkage may occur as fibrous tissue maturation progresses.

The occurrence of serious infection in the free cutis graft was small (3 per cent). This finding is particularly significant in view of the fact that no special effort was made to reduce the bacterial flora of the skin. In addition, the animals were given only minimal antibiotic protection.

In a recent publication of the bacterial flora of the normal human skin Propionibacterium acnes was by far the most numerous organism in most individuals. Micrococcus epidermidis, Staphylococcus albus, and Micrococcus candidus were also regularly present. It was shown that the sebaceous glands were the chief site of bacterial growth. Sweating caused only a minor transient increase in skin flora. Therefore, the experiments reported with cutis grafts in dogs, an animal essentially without sweat blands, appear to be valid for humans. One might anticipate less difficulty with the problem of infection in humans, for cultures obtained in some animals with deep infections revealed enteric organisms. One might expect coliform and related organisms to be regular inhabitants of the skin and sebaceous glands of dogs. Although the organisms found in normal human skin are regarded as nonpathogenic, some are probably sensitive to certain of the antibiotics. It is planned in a future study to explore the sensitivity of human skin organisms to the various antibiotics.

Adhesions between the diaphragmatic surface of the pleural and peritoneal organs and the graft occur regularly. The resulting fixation produces no apparent physical disability. The circulation through these adhesions and the periphery of the diaphragmatic defect accounts for the consistent survival (96.7 per cent) of these large grafts.

The histopathology of the cutis graft was of considerable interest. In general, the hair follicles underwent cystic degeneration by 1 month; granulomatous areas containing lymphocytes, plasma cells, and foreign body giant cells persisted throughout the 4 month period. Microscopic "abscesses," at the site of the sebaceous glands persisted over a month. A detailed presentation of these changes will be the subject of a subsequent publication (6).

Architectural changes within the viscera adhered to the graft appeared only in the lung. Thickening of the pleura was present routinely. Pneumonitis or rarely "alveolar" pneumonia in the immediate vicinity of the graft was observed microscopically extending a few millimeters into the parenchyma.

CONCLUSIONS

1. Free cutis grafts provide a satisfactory diaphragmatic substitute. The pedicle cutis

graft offers no advantages and has several major disadvantages.

2. Partial separation of the free cutis graft did not occur; deep infection occurred in 3 per cent (1 dog). Minimal antibiotic protection was used.

3. Approximately 30 per cent shrinkage of the graft occurred in 4 months.

4. Bilateral diaphragmatic hemiresections (90 per cent of the total, bilateral, diaphragm) followed by free cutis substitution have been successful. No apparent physical disability of the animals was observed.

5. The major microscopic changes occurring in the cutis graft when utilized within a body cavity consist of cystic degeneration of the hair follicles, the presence of minute granulomatous areas of chronic inflammatory cells, and scattered microscopic "abscesses."

6. In the human patient with the adequate protection of antibiotics, and without the severe skin contamination observed in the experimental animal, the incidence of infection should be even less than the experience herein recorded with dogs.

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THE NEED FOR ADEQUATE AFTERCARE IN COMPLETE REHABILITATION OF THE DISABLED

ALEXANDER P. AITKEN, M.D., F.A.C.S., Brookline, Massachusetts

THE total number of industrial accidents which occurred in 1950 in the United States reached a figure of 1,952,000. Of this number 15,500 ended fatally while 84,900 ended in some degree of permanent disability.[1] It has been estimated that there are 2,000,000 persons in this country disabled either as the result of injury or disease who could and should be rehabilitated and placed in employment.[2] The entire problem of restoration of the injured worker to gainful employment including adequate medical care and vocational rehabilitation has become of vital importance not only to the Federal Government but to many powerful lay groups including organized labor and management. Although such restoration is primarily a medical problem and of vital concern to our profession, the true significance of this subject and the consequent solution of the problems presented have passed unnoticed by us.

There can be no denial that medicine as practiced at the present time in these United States has no equal throughout the world. The surgical handling of the case of acute trauma has reached a plane never before attained. This is due in part to the stress laid upon the importance of adequate first aid, the transportation of the injured, and the standardization of hospitals. It is due in large measure to the tremendous strides made in the surgical handling of the various traumatic lesions aided immeasurably by the discovery of the antibiotic drugs. The more recent developments made in the treatment of open wounds, in the field of plastic, hand, and fracture surgery suffice to indicate the progress made in other fields.

Unfortunately rehabilitation of the patient does not end with the healing of surgical wounds. Much more must be done, far more than is now appreciated by the medical profession in general. Failure to recognize the importance of adequate surgical or medical aftercare represents one of the major weaknesses of modern-day medicine. One of the best examples of the deficiency of present-day aftercare is the treatment given the average civilian amputee. Legion are the surgeons who can perform excellent amputations. Pitifully few are those who can supervise the procurement and fitting of an efficiently functioning prosthesis. This problem, left usually to the prosthesis maker whose primary interest is the sale of apparatus, is truly a medical one and should be so handled. What may be said of the deficiencies in the handling of the amputee can also be said to some extent about the aftercare of the average case of trauma.

Failure to comprehend the value of adequate aftercare stems largely from our basic training. The modern medical school devotes its time largely to the field of diagnosis and treatment of the acute lesion. Rarely is any instruction given, either in the field of medicine or surgery, to the treatment of the individual once the emergency has passed. The training of interns and residents, again, is one of instruction in treatment while the patient is hospitalized. Once the patient leaves the hospital he is on his own to rehabilitate himself as best he can. Postgraduate training as presented by medical schools, hospital staffs, and medical societies again stresses the importance of accurate diagnosis and technique in the management of the acute condition. Nowhere does there appear to be a profound appreciation of the patient's overall problem. How many patients discharged from the hospital as "cured" really are cured in the true sense of the word?

CONCEPT OF REHABILITATION

What is meant by rehabilitation? For every group interested in this field there appears to

[1]Bureau of Labor Statistics, U. S. Department of Labor.
[2]Report to the Chairman, Manpower Policy Committee, Office of Defense Mobilization, by the Task Force on the Handicapped.

be a different concept. To many, particularly the lay public, the term rehabilitation has become synonymous with physical medicine embracing physical, occupational, and recreational therapy. To others it means vocational training; while to still others the problem is a psychological one to be treated in the field of psychiatry. These specialties, invaluable to restoration, are but adjuncts in the comprehensive concept of rehabilitation. Rehabilitation means the restoration of the patient to self-sufficiency or to gainful employment at his highest attainable skill in the shortest possible period of time.

On what then does successful rehabilitation depend? It depends first upon the extent and severity of the accident. Second, it depends upon the competence of handling of the acute emergency. If adequate repair or reconstruction has not been accomplished, then function will never return to the injured part despite the efficiency of postoperative care. To insure the maximum restoration of function, physicians must recognize the limitations of their own ability and not hesitate to call in those more capable of treating the emergency. Adequate treatment of the acute lesion is perhaps the most important factor in successful rehabilitation.

Ranking next in importance is a third factor, that of adequate treatment in the convalescent period. Physicians in general and surgeons in particular must learn the direct effect such treatment has upon the surgical end result. The utilization of the minimum amount of splinting to permit early active use of uninvolved muscles and joints is a factor not generally appreciated and one which should be corrected by competent instruction in medical school, hospital, and postgraduate teaching.

Another equally important factor is the use of exercise, not only for the injured part, but for the body as a whole. How many excellent reductions of a Colles' fracture end disastrously because of swollen and stiff hands or frozen shoulders due to lack of early active exercise of these uninvolved joints? How many patients confined to bed for weeks are unnecessarily retarded in their ambulation because their general body tone has been allowed to deteriorate due to lack of exercise which could be given during enforced recumbency? All patients, particularly those who are ambulatory and under domiciliary care must be placed on those exercises which will restore function to a maximum degree in the shortest possible time. These exercises should include those obtained through work therapy. The patient must be given specific instructions as to how, when, and why these exercises should be done. This means scrupulous attention to detail on the part of the physician. Unfortunately many physicians feel they have no time for such detailed supervision. This, however, is just as important an aspect of adequate care as is the treatment of the acute emergency and is, therefore, a necessary task which we must not shirk.

In many instances physical facilities may not be available either in the doctor's office or in the patient's home, or the patient may be the type who will do nothing for himself unless constantly supervised. In such cases, the patient should be sent, if possible, to a special center where such facilities and supervision can be given. There is a great demand on the part of many lay groups for the establishment of special rehabilitation centers throughout the country. The need for such facilities, particularly for the seriously injured, cannot be denied. The real need, however, is for the doctor to realize that once he has undertaken the care of a patient, his obligations to that patient do not cease until he has been restored to as near normal as is humanly possible. To that end the doctor must learn how to administer the most efficient type of aftercare or to utilize the services of those individuals or institutions capable of so doing. There is an excellent opportunity for much useful work to be done by our educational institutions in offering courses to the medical profession in the use of exercise and work therapy. Much can be done in this respect to improve the end results and to truly rehabilitate the patients whether they suffer from either surgical or medical lesions.

The fourth factor which plays a profound part in restoration of injured individuals is the recognition of intercurrent medical diseases. Diabetes, nephritis, anemia, and vascular

lesions, to name a few, may do much to retard convalescence. Oftentimes surgeons are apt to overlook such a medical condition. Any surgical case which fails to make satisfactory progress should be examined by a competent physician to rule out such possible complications.

The fifth factor which all too frequently plays a decisive role in rehabilitation is the development of the psychological complications of trauma. In some instances psychoneurosis or mental depression may develop to such a degree as to require the aid of a psychiatrist. In many instances, however, the depression is brought on by a feeling of insecurity. Loss of income, caused by injury, may have a profound effect not only upon the patient but his entire family. Worry over inability to meet current expenses, to keep a child in school, or to meet mortgage payments is a major factor in the production of such a depression which, in turn, has a deleterious effect upon the patient's recovery. This may not seem to be of concern to the doctor but it is, nevertheless, a complication of trauma. If the physician has the confidence of the patient, a few moments of frank discussion may do much to relieve the patient of his anxiety. Not infrequently the physician may have knowledge of how financial aid might be obtained and thus afford the patient some relief from his apprehension.

Often depression may be due to fear of loss of employment either through the loss of a job, or inability to return to his regular work. If the physician is aware of this fear, a telephone call to the employer will often result in a promise of work. In cases in which an individual cannot return to his regular work and in which jobs are frozen by seniority rights, discussion with management and union representatives may be productive of a promise of work within the patient's capacity.

It may seem to many that this type of social service is not a duty required of the attending physician. However, we must remember we are doctors and that we are treating sick patients. The psychological effects of trauma are as real and as important to the patient and his recovery as any surgical complication. It is our duty to see the patient through his difficulties, mental as well as physical, and to give him any and every assistance possible. Doctors should be more than mere technicians; patients look to them for physical, psychological, and at times, even spiritual help. The treatment of such psychological complications is clearly a responsibility of the medical profession.

Frequently a patient is so badly injured that he can never return to his regular work. This fact is often obvious to the surgeon within a short time after the accident. If such an appraisal is made, then the patient and his family should be made aware of this probability. Efforts should then be made to contact an agency interested in vocational training. The sooner such training is started, the sooner the patient will return to work at his highest attainable skill. It is not fair to the individual to allow him to entertain the hope that he can eventually return to his regular work when he obviously cannot do so.

In 1951, 4,430 persons injured on the job while under workmen's compensation were returned to work under the federal-state program of vocational rehabilitation.[1] The average time lag between injury and referral to the vocational rehabilitation agencies, however, was 7 years. The economic loss to the patient and to the community in these cases was tremendous. There are undoubtedly many factors which operate to produce such prolonged disability but in some of the cases restoration could conceivably have been hastened by more careful medical supervision.

There are numerous groups and agencies, both public and private, interested in the rehabilitation of the disabled. They ask only prompt referral of such individuals to them. No one is in a better position to see that such facilities are utilized than the attending physician. He knows the patient and his needs better than anyone else and should see that such assistance is given. Most physicians, however, are unaware of the existence of such agencies due in large measure to the failure of these groups to present their function and facilities to the medical profession. One does not reach the average physician through the

[1]Report to the Chairman, Manpower Policy Committee, Office of Defense Mobilization, by the Task Force on the Handicapped.

medium of pamphlets. Such literature is apt to be afforded the same treatment as is given to much of the useless literature that is piled daily upon the doctor's desk. It would seem that the medical societies could do much to familiarize their members with the existence and function of these institutions and how their services may be obtained.

CONCLUSIONS

This over-all concept of rehabilitation should make it clear to all that rehabilitation is purely a medical problem and must be handled by the physician as such. The doctor must realize that his duty to his patient is not ended until that patient has returned to work at his highest attainable skill. Rehabilitation and restoration to gainful employment of the injured worker must begin with first aid and continue through the period of disability. In order for a physician to carry out this responsibility, it is essential for him to recognize the total medical problem of the patient in addition to his injury, as well as his personal problems. The physician must bring to bear on these problems all the skills and disciplines that science and society can offer, and utilize all community resources which can assist him in the accomplishment of these objectives.[1]

In the successful rehabilitation of any individual there must be a hand at the helm to steer a course for the disabled person. There must be no drifting. A course must be plotted to insure continuous supervised treatment. Although such treatment may be rendered in various ways by numerous individuals, the guiding hand must be the one most capable of so doing, that of the attending physician.

Failure of the medical profession to adopt an over-all concept of rehabilitation, and the responsibilities inherent therein, may well see pressure applied by numerous powerful lay groups upon the government for the solution through legislation of this entire problem. These groups, already highly critical of the medical profession for its failure to keep pace with the numerous social changes, will become more potent the longer we fail to recognize and solve those problems which are directly our concern.

[1]Principles for rehabilitation of injured worker established. Bull. Am. Coll. Surg., July-August, 1952, Vol. 37, No. 2.

DIAGNOSIS, TREATMENT, AND PROPHYLAXIS OF POTASSIUM DEFICIENCY IN SURGICAL PATIENTS

Analysis of 404 Cases

HYMAN S. LANS, M.D., IRVING F. STEIN, JR., Ph.D., M.D., and
KARL A. MEYER, M.D., F.A.C.S., Chicago, Illinois

REPORTS have appeared during the past several years concerning the occurrence of potassium deficiency in many clinical conditions including: infantile diarrhea, alkalosis due to vomiting, the recovery phase of diabetic acidosis, periodic paralysis, bulbar poliomyelitis, and, following the administration of adrenocorticotrophic hormone, cortisone or testosterone. Randall and associates have shown that many of the conditions reported as leading to potassium deficit often exist in surgical patients and case reports have appeared in the recent literature.

Most reports of potassium deficiency have dealt with small groups of carefully selected surgical patients. The present report concerns our experiences in the diagnosis, treatment, and prophylaxis of potassium deficiency based upon an analysis of 404 surgical patients. These patients were studied and treated at the Cook County Hospital during a 12 month period. Long existing illness upon admission and advanced disease in many patients probably account for the large group of cases occurring in a 1 year period at this charity institution. During the same year we observed a relatively much smaller number of cases in our private patients.

METHODS

A research group was established to study electrolyte abnormalities in surgical patients. Patients were studied when there were any etiologic factors present which could lead to electrolyte imbalance and particularly potassium deficiency. Other patients were brought to our attention for treatment of existing electrolyte abnormalities. Another group of patients undergoing major surgery were studied preoperatively and postoperatively. Treatment of potassium deficiency was directed by the research group.

Laboratory studies routinely performed included: serum sodium, chloride, potassium, carbon dioxide combining power, blood nonprotein nitrogen, and creatinine. Determinations of electrolytes in urine and gastrointestinal secretions were performed frequently. Sodium and potassium concentrations were determined on a flame photometer with lithium as an internal standard (1). Standard laboratory procedures were used for other determinations.

Blood was collected in the morning under fasting conditions, except in emergencies. The daily fluid and electrolyte therapy were based on the laboratory determinations performed the same morning, and response to treatment was followed by daily (or more frequent) blood chemistries on each patient.

CLINICAL MATERIAL AND TYPES OF POTASSIUM DEFICIENCY

Deficiency with hypopotassemia. We consider that the normal range of serum potassium is from 4.0 to 5.6 milliequivalents per liter (5). A hypopotassemia (hypokaliemia) usually indicates a potassium deficiency, although the intensity of that deficiency cannot always be diagnosed from the serum level itself. Three hundred and nine patients in this series had a serum potassium below 3.7 milliequivalents per liter and so were considered to have definite deficits of body potassium.

From the Departments of Surgery of the Cook County Hospital and Northwestern University Medical School and from the Hektoen Institute for Medical Research of the Cook County Hospital.

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TABLE I.—TWO HUNDRED FORTY-ONE PATIENTS WITH PREOPERATIVE HYPOPOTASSEMIA

Diagnosis	No. of cases
Small bowel obstruction	65
Pyloric obstruction	34
Peptic ulcer	26
Gastric carcinoma	8
Large bowel obstruction	26
Gonorrheal peritonitis	15
Small bowel fistula	13
Perforated peptic ulcer	13
Pancreatitis	12
Esophageal carcinoma	10
Acute cholecytitis	10
Bleeding peptic ulcer	6
Obstructive jaundice	6
Ruptured tubo-ovarian abscess	6
Burns	5
Appendiceal abscess	5
Tetanus	4
Large bowel fistula	4
Diverticulitis colon	3
Gastric carcinoma	2
Regional ileitis	1
Ulcerative colitis	1

Preoperative patients.—Two hundred and forty-one patients had a potassium deficiency preoperatively. These cases are listed according to diagnosis in Table I.

One hundred and sixty patients had a hypopotassemia on admission to the hospital. The majority of these patients had little or no oral intake of food for at least 3 days preceding admission. Loss of gastrointestinal fluids and electrolytes was prominent in many patients because of vomiting (75 patients), diarrhea (6 patients), or gastrointestinal fistulas (17 patients). Acute blood loss of marked degree was present in 5 patients.

Fourteen patients with a normal serum potassium on admission were found to have a hypopotassemia following correction of severe dehydration with parenteral saline and glucose solutions.

Sixty-seven patients had a hypopotassemia after varying periods (4 days to 5 weeks) of hospitalization. All these patients had been on potassium-free intravenous fluids since admission. All but 9 had been on gastric suction-drainage since shortly after admission.

In the above groups, factors which could have led to a potassium deficit were present for less than 5 days in 170 patients, for 5 to 10 days in 23 patients, for 10 to 20 days in 19 patients and for longer than 20 days in 29 patients.

Postoperative patients.—Sixty-nine patients developed a persistent hypopotassemia postoperatively. Abnormally low serum potassium levels were present for at least 96 hours postoperatively and all the patients exhibited symptoms suggestive of potassium deficit. The surgical procedures performed in these cases are listed in Table II. Extensive operations were performed in the majority of these patients (36 for gastrointestinal carcinoma). Gastric suction was used postoperatively in all cases; 10 patients had abnormal losses of gastrointestinal fluids through a fistula, such as a choledochostomy, cecostomy, or colostomy.

Deficiency with normal serum potassium. It is known that a low concentration of serum potassium may not be found in all cases in which a low intracellular potassium exists. The concentration of potassium in the serum represents the result of several processes; namely, the ingestion of potassium, its liberation by tissues and excretion in the urine and feces. Studies showing a negative potassium balance or a urinary excretion of potassium in excess of 2.75 mEq. for every gram of nitrogen excreted are direct evidence of a deficit of potassium. Presumptive evidence is present when administration of potassium results in a disappearance of clinical manifestations attributable to potassium deficiency. Moore has termed this "chronic intracellular potassium deficit" and it occurs when starvation is present and a normal extracellular concentration of intracellular electrolytes is maintained.

Eleven patients with chronic illnesses in our series had a normal serum potassium, but exhibited symptoms of hypopotassemia such as extreme muscular weakness and lethargy. These patients had a prolonged poor food intake and showed evidence of marked weight loss and malnutrition. Gastrointestinal carcinoma was present in 8 patients, fractures of the femur in 2, and a severe body burn in 1. Supplemental potassium intake resulted in improvement in muscular strength and mental attitude in 4 of these patients. These 4 patients probably had chronic intracellular potassium deficits.

Transient postoperative decrease in serum potassium. A fall in serum potassium of transient nature frequently occurs following ex-

tensive surgery. This alteration is due to an increased renal excretion of potassium and is part of the normal reaction of the body to stress. The serum potassium returns to normal without treatment if other etiologic factors are not present.

In our series 15 patients with a normal serum potassium preoperatively were found to have a hypopotassemia on the first postoperative day. However, the serum potassium returned to normal within 48 hours in 9 patients and within 72 hours in the other 6 patients, without treatment or oral food intake. The clinical diagnosis and serum potassium level of each case is listed in Table III.

PATHOGENESIS OF POTASSIUM DEFICIENCY IN SURGICAL PATIENTS

The pathogenic factors of the patients in this study were classified as: (1) limitation of potassium intake, and (2) loss of potassium from the body.

Limitation of potassium intake. The average normal intake of dietary potassium is from 3 to 4 grams or 75 to 100 milliequivalents per day. There is no storage of potassium in the normal adult; instead, the excess intake is excreted in the urine. Howard and Carey have demonstrated the importance of prior malnutrition as a factor favoring the development of postoperative potassium deficits. In malnutrition or dehydration, cellular breakdown occurs with resultant release of potassium from the cell. This potassium is excreted in the urine. Prolonged use of parenteral fluids is often necessary in surgical patients to provide nutrition and prevent dehydration.

TABLE II.—SURGICAL PROCEDURES PERFORMED IN 69 PATIENTS DEVELOPING POTASSIUM DEFICIENCY POSTOPERATIVELY

Surgical procedure	No. of cases
Subtotal gastrectomy	16
Hemicolectomy	15
Esophagectomy	6
Cecostomy	6
Total gastrectomy	4
Choledochostomy with cholecystectomy	4
Abdominoperineal resection	4
Small bowel resection	4
Colostomy	3
Abdominoenteric fistulectomy	3
Pancreaticoduodenectomy	2
Abdominal hysterectomy	2

TABLE III.—PATIENTS WITH POSTOPERATIVE HYPOPOTASSEMIA WHICH RETURNED TO NORMAL WITHOUT TREATMENT OR ORAL FOOD INTAKE

Diagnosis	Preoperative serum K mEq./L.	Serum potassium, first postoperative day mEq./L.
Subtotal gastrectomy	4.2	3.6*
Subtotal gastrectomy	4.6	3.8**
Subtotal gastrectomy	5.1	3.5**
Hemicolectomy	4.4	3.7*
Hemicolectomy	5.0	3.5**
Hemicolectomy	4.4	3.2*
Pneumonectomy	4.3	3.4*
Esophagectomy	5.6	3.7**
Cholecystectomy	5.3	3.4**
Cholecystectomy	4.0	3.7**
Cholecystectomy	4.4	3.5**
Hysterectomy	4.6	3.5**
Small bowel resection	5.8	3.7*
Exploratory laparotomy	4.2	3.8*
Ventral herniorrhaphy	4.6	3.8**

*Returned to normal within 72 hours
**Returned to normal within 48 hours

The solutions usually used contain little or no potassium. Therefore, patients sustained on these solutions alone receive an inadequate intake of potassium.

An inadequate intake of potassium was present in the majority of the patients in this study and the administration of potassium-free parenteral solutions often increased an existing hypopotassemia.

Loss of potassium from the body. Excretion of potassium.—The kidney is the principal route for potassium excretion. The usual urinary excretion of potassium is 2 to 3 grams per day. Normally, less than 10 per cent of ingested potassium is found in the stool and a considerably less amount in the sweat. Renal excretion of potassium occurs by both glomerular filtration and tubular excretion. Potassium balance is therefore normally maintained by the processes of glomerular filtration, tubular excretion, and tubular reabsorption. Although the daily total excretion of potassium may be decreased, renal losses often continue even during periods of marked potassium deficiency.

The renal mechanisms for potassium excretion are influenced by many extrarenal factors, including disturbances in acid-based balance, infusions of parenteral solutions and body stress. Changes in acid-bases balance occurred in many patients in this study. Pa-

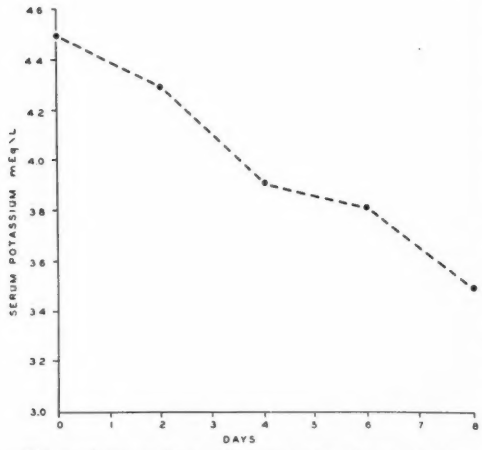

Fig. 1. Average changes in serum potassium concentration of 9 subjects on potassium-free intravenous fluids. Subjects received an average of 2,000 cubic centimeters of 5 per cent dextrose and 1,000 cubic centimeters of 0.9 per cent sodium chloride solutions daily.

tients who lost a large volume of gastric juice developed alkalosis while others with losses of pancreatic, biliary, and intestinal secretions often developed an acidosis. Darrow has demonstrated in experimental animals that induced disturbances in acid-base equilibrium result in an increased urinary excretion of potassium and a lowered cell potassium. Infusions of large amounts of parenteral solutions may result in diuresis and increase the renal loss of potassium. Large infusions, oral or parenteral, of sodium salts induce losses of intracellular potassium. Therefore, in patients with potassium deficiency the intake of sodium should not be excessive. The effect of potassium-free intravenous fluids on serum potassium in 9 subjects is illustrated by Figure 1. The adrenal cortex regulates the distribution and excretion of potassium. There is a close resemblance between the clinical and electrolyte changes seen postoperatively and those described following administration of ACTH or cortisone and in some cases of Cushing's syndrome. Surgical trauma causes an increased production of adrenocroticotrophic hormone by the pituitary body which stimulates the adrenal cortex to secrete one or more of its hormones. These hormones have specific effects on sodium and potassium excretion and this stress response results in sodium retention and increased renal loss of potassium.

Loss of gastrointestinal secretions.—In addition to the loss of potassium through increased renal excretion, many patients in this study had an additional source of potassium loss through vomiting, diarrhea, fistulas, or the use of gastrointestinal suction-drainage. Gastric juice has been reported to contain as much as 40 milliequivalents of potassium per liter and intestinal juices, usually 6 to 8 milliequivalents per liter. Determinations of the electrolyte composition of various gastrointes-

TABLE IV.—AVERAGE VALUES AND RANGES OF SODIUM, POTASSIUM, AND CHLORIDE IONS IN GASTROINTESTINAL FLUIDS

	No. of specimens	No. of patients	Sodium mEq./L.	Chloride mEq./L.	Potassium mEq./L.
Gastric juice* (free HCl)	76	26	56 (21–82)	126 (76–157)	12.6 (6.5–26.0)
Gastric juice* (no free HCl)	65	19	65 (24–126)	72 (36–122)	11.2 (4.8–25.6)
Bile	48	27	151 (137–181)	103 (94–126)	5.2 (3.0–9.2)
Mixture of gastrointestinal secretions†	64	41	113 (46–117)	104 (24–127)	4.6 (3.2–16.1)
Small bowel secretions‡	12	3	121 (88–146)	98 (91–119)	9.6 (4.8–13.6)
Duodenal fistula	19	2	96 (75–119)	61 (52–82)	8.1 (7.2–13.7)
Ileal fistula	12	3	132 (129–138)	68 (35–76)	6.2 (4.6–9.1)
Cecostomy drainage	17	6	69 (52–74)	38 (26–49)	8.2 (4.6–14.0)

*Pyloric obstruction †Gastric suction ‡Intestinal suction

tinal secretions were performed on 313 specimens obtained from 126 subjects. The average values and ranges of the concentrations of sodium, chloride, and potassium ions are listed in Table IV. Potassium was found to be present in relatively high concentration in all types of gastrointestinal secretions. The largest amount of potassium was present in gastric juice in concentrations 2 to 5 times greater than in serum. The other gastrointestinal secretions contained potassium in concentrations equal to or twice that of serum.

Normally the potassium content of the gastrointestinal secretions is reabsorbed. However, patients with a daily loss of 3 to 4 liters of gastrointestinal fluids may lose as much as 81 to 108 milliequivalents of potassium per day (equivalent to the potassium content of 6 to 8 grams of potassium chloride).[1] If this loss of potassium continues for several days, a quantity of potassium greater than that found in the serum and extracellular fluids will be lost from the body. This loss of potassium contributes significantly to the development of potassium deficit. Figure 2 illustrates that the use of gastrointestinal suction-drainage, without adequate intake of potassium, often leads to potassium deficit after 4 or 5 days.

Loss of potassium through hemorrhage.—The potassium content of human whole blood averages 45.5 milliequivalents per liter. Patients with severe hematemesis or melena lose large amounts of potassium as a result of blood loss and the increased renal excretion of potassium invoked in response to the stress of hemorrhage.

CLINICAL MANIFESTATIONS OF POTASSIUM DEFICIENCY

The relative frequency of the signs and symptoms exhibited by patients in this study are listed in Table V. There was no correlation between serum potassium levels and clinical findings; 4 patients with serum potassium below 2.9 milliequivalents per liter were asymptomatic, while other patients with serum potassium of 3.6 milliequivalents per liter had definite symptoms of potassium deficiency. Hypopotassemia indicates a marked intracellular deficit of potassium and the clinical findings probably are a result of altered cellular potassium rather than of lowered serum potassium.

[1]A solution containing 1 gram of KCl contains 13.5 mEq. of potassium. Therefore, 81 divided by 13.5 gives 6 grams of KCl.

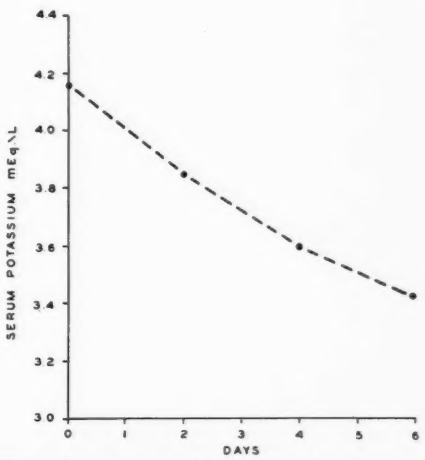

Fig. 2. Average values of serum potassium concentration of 17 subjects on Levin suction. All subjects had a normal serum potassium before continuous Levin suction was started. Daily fluid intake consisted of 2,000 cubic centimeters of 5 per cent dextrose and 1,000 cubic centimeters of 0.9 per cent sodium chloride solutions.

Muscular weakness, gradual in onset, and of increasing severity, was the most impressive clinical finding. The paresis usually involved muscles of the extremities and trunk but rarely muscles supplied by cranial nerves.

TABLE V.—RELATIVE FREQUENCY OF SIGNS AND SYMPTOMS IN 322 SURGICAL PATIENTS WITH POTASSIUM DEFICIENCY

Usually present
Anorexia and nausea
Muscular weakness (often of marked degree and with loss of deep tendon reflexes)
Mental depression: lethargy and apathy; drowsiness
Frequently present
Mental confusion
Shallow respirations
Abdominal distention and paralytic ileus
Irregular pulse and fall in blood pressure
Infrequent
Complete muscular paralysis
Delirium and hallucinations
Not observed
Complete respiratory paralysis
Cranial nerve paralysis

Diminution or absence of deep tendon reflexes was common. An acute syndrome of skeletal muscular paralysis has been described by Randall. This was observed in only 6 of our cases and consisted of complete paralysis of the lower extremities. The majority of patients exhibited varying degrees of listlessness and mental depression. Anorexia was also a frequent finding and served to increase the existing potassium deficit. Respiratory difficulty, apparently due to weakness or paralysis of the intercostal muscles or diaphragm, frequently occurred and was characterized by rapid, shallow, gasping type of respiration and was occasionally accompanied by cyanosis. Shallow infrequent respirations may be important in the development of postoperative pulmonary complications. The possibility of death from respiratory paralysis due to potassium deficit has been emphasized in a number of reports. Actual respiratory paralysis was not observed in any of our surgical patients.

Abdominal distention was present in 31 patients without bowel obstruction. Eighteen patients had a silent abdomen and in the other 13 bowel sounds were markedly diminished. Administration of potassium chloride resulted in a rapid diminution of distention and a return of bowel sounds and spontaneous passage of flatus. Experimental studies have demonstrated that induced potassium deficit results in paralysis of gastrointestinal musculature. Ileus resulting from potassium deficiency may increase postoperative complications by contributing to fluid and electrolyte depletion and malnutrition. Six patients had prolonged periods of delayed gastric emptying and vomiting following subtotal gastrectomy; correction of their potassium deficit was followed by normal gastric emptying and cessation of vomiting.

Alterations in cardiac function are common in severe potassium deficiency. Clinically, cardiac arrhythmia, fall in diastolic pressure with increased pulse pressure, the development of systolic murmur, and cardiac dilatation were observed. Cardiac arrest occurred during surgery in 4 patients, of which 2 recovered. Three of these patients had severe potassium deficits which were treated with intravenous potassium chloride only until the serum potassium was normal. The other patient was found to have a serum potassium of 3.1 milliequivalents per liter and a serum calcium of 10.2 milligrams per cent ½ hour after restoration of cardiac activity. This patient had also been inadequately treated for a potassium deficiency preoperatively. Myocardial necrosis has been found in animals with experimentally induced potassium deficits. Cardiac arrest in the first 3 patients may have been due to such myocardial lesions and in the other patient to increased cardiac irritability due to hypopotassemia with a normal serum calcium. It is suggested that potassium deficiency may contribute to cardiac arrest and, therefore, when possible surgery should not be performed unless potassium therapy has been administered for a sufficient time to restore intracellular potassium.

The electrocardiographic changes in hypopotassemia have received considerable attention. With moderate decrease in serum potassium the T wave becomes slightly lower but broader with lengthening of the Q-T interval. As the serum concentration is further decreased there is first a lowering or inversion of the T wave and then a sagging of the ST segment. A U wave may be noted. Electrocardiographic studies were conducted on 61 patients. Changes ascribable to alteration of serum potassium were present in the tracings of 39 patients. The electrocardiographic changes did not appear consistently at specific serum potassium levels. Furthermore, in 7 patients with serum potassium of 2.9 milliequivalents per liter or less a normal tracing was obtained. Therefore, a normal electrocardiogram does not rule out the presence of a potassium deficiency. However, electrocardiography is an excellent method of determining the development of hyperpotassemia during rapid intravenous potassium salt administration. Potassium can be safely given until the T wave becomes high and peaked.

Evisceration occurred between the seventh and ninth postoperative days in 2 patients who had undergone subtotal gastrectomy and in 1 patient who had a hemicolectomy. Total serum protein and albumin were normal; hypopotassemia was present in all 3 patients. Low intracellular potassium may be an impor-

tant factor leading to postoperative wound dehiscence or evisceration because potassium is necessary for tissue regeneration and potassium deficiency may actually lead to tissue breakdown.

Six patients with a cecostomy rapidly developed marked hypopotassemia when the cecostomy was opened. In addition to a general diet, intravenous potassium therapy was necessary during the first 2 postoperative weeks to maintain a normal serum potassium in these patients. Thirteen patients with a transverse colostomy developed only a slight decrease in serum potassium which was corrected by oral food intake alone.

Hyperpotassemia has been cited as an important lethal factor in intestinal obstruction. An abnormally high serum potassium concentration was not found in 109 cases of large or small bowel obstruction. Hypopotassemia was present in 91 of these patients.

Sixty-four patients had an alkalosis evidenced by a serum carbon dioxide combining power greater than 75 volumes per cent (33 mEq./L.). An associated hypochloremia was present in 41 of these patients. Twenty-one patients had been treated with sodium chloride alone. The alkalosis was resistant to or was made worse by this therapy in 17 patients. Administration of potassium chloride intravenously to the remaining 60 patients resulted in a prompt correction of the alkalosis and hypochloremia. In 9 patients with a serum carbon dioxide combining power greater than 90 volumes per cent (40 mEq./L.), tetany was not observed until the serum potassium level was elevated above 3.6 milliequivalents per liter. Prompt cessation of tetany followed the intravenous administration of calcium chloride or gluconate. Clinical potassium deficit with hypochloremic alkalosis has previously been reported. Howard and Carey have demonstrated that large amounts of sodium chloride or ammonium chloride are ineffective in treating hypochloremic alkalosis if a coexisting potassium deficit is not corrected. Darrow has shown in potassium deficient rats that alkalosis and potassium deficit can be accounted for by a diuresis resulting in a relatively larger renal loss of sodium than of chloride.

THERAPY OF POTASSIUM DEFICIENCY

Administration of potassium chloride intravenously was followed in most of the 314 cases by prompt and often striking clinical improvement. In 23 cases this appeared to be an emergency lifesaving measure. We have previously observed many patients with intestinal fistulas, intestinal obstruction, or paralytic ileus, who expired before the recognition and treatment of potassium deficiency. The marked clinical improvement in the clinical status of the patients in this series was always accompanied by an elevation of serum potassium, but it was not always necessary to restore the concentration to normal to obtain this result. This has been observed by other workers. The number of days of intravenous treatment required to restore the serum potassium to normal averaged 3 days. Hypopotassemia reappeared in a day or two, if administration of potassium was stopped as soon as the serum potassium returned to normal. When potassium was administered for 4 days after restoration of normal serum potassium, recurrence of hypopotassemia was not observed provided etiologic factors of potassium deficit were no longer operative. Thirteen patients with chronic potassium deficits required supplemental administration of potassium salts for longer than 3 weeks before the serum potassium remained normal.

Types of solutions and methods of administration. Administration of potassium salts must be done cautiously and only under proper laboratory and clinical supervision. Concentrated solutions or rapid administration may lead to a high serum level of potassium, which is toxic to the heart and which has been reported as causing heart block and cardiac arrest. Therefore, although we have used solutions containing as much as 6 grams of potassium chloride in a liter, we recommend for general use a solution containing 3 grams of potassium chloride (40 mEq. of potassium[1]) in a liter of intravenous or subcutaneous fluids. Such solutions should be administered not faster than 120 to 180 drops (8 to 12 c.c.) per minute.

[1]A solution of 1 gram of potassium chloride in a liter of distilled water contains 13.51 mEq. of potassium and 13.51 mEq. of chloride.

Potassium must be administered very cautiously to patients with renal insufficiency or dehydration. In nephritis there may be a diminution of glomerular filtration, but tubular excretion may be almost normal. As a consequence, if urine output is adequate potassium may be excreted in relatively normal amounts and a potassium deficiency will develop when intake of potassium is low. However, if oliguria occurs and is associated with tissue breakdown the serum potassium level may be high. It is, therefore, recommended that in patients with kidney disease potassium *not* be administered unless a low serum potassium level exists. Potassium should also be withheld during the first 24 postoperative hours, unless *a hypopotassemia exists*, because renal function is often depressed and a hyperpotassemia may result. Similarly patients with normal kidneys but with severe dehydration may have a temporary oliguria and hemoconcentration with a resultant normal or high serum potassium. The true state of the serum potassium in these patients can usually be recognized following hydration with saline and/or glucose solutions. As shown by our previous studies (4), an elevation of the blood nonprotein nitrogen is not a contraindication to the administration of potassium-containing solutions as long as a hypopotassemia is present.

Approximately 8,000 liters of intravenous and subcutaneous fluids containing potassium chloride were administered during this study. No complications were observed during or following this therapy.

Experimental studies in animals have demonstrated the importance of potassium as a dietary essential in the process of tissue protein regeneration. Marked losses of intracellular potassium and protein are often concomitantly present since both deficiencies have many common etiologic factors. Therefore, we believe that the addition of potassium salts to protein hydrolysates provides a solution well-suited for use in surgical patients, as it provides amino acids and potassium, both of which are essential for the effective reconstruction of intracellular protein. We used such solutions in approximately 25 per cent of our cases.

Daily therapy.—The following routine is suggested as a basis for the treatment of potassium deficiency in surgical patients. Accurate treatment depends on daily blood chemistries including serum sodium, potassium, chloride, and carbon dioxide. Any suggested dosage must be adjusted to the needs and responses of the individual patient.

When a potassium deficiency is found to be present, 3 grams of potassium chloride are added to each liter of intravenous or subcutaneous fluids so that the patient will receive 9 to 15 grams of potassium chloride (121 to 202 mEq. of potassium) per day. This treatment is continued until the serum potassium has been normal for 24 hours, after which 6 grams of potassium chloride are administered daily to patients who are not on gastrointestinal suction drainage. If losses of gastrointestinal secretions through suction drainage or fistulas are present, in addition to the above intake of 6 grams, each liter of gastrointestinal secretions lost in a 24 hour period is replaced parenterally with a liter of dextrose and sodium chloride solution (0.45 per cent sodium chloride and 2.5 per cent dextrose) to which is added 3 grams of potassium chloride. The continued provision of potassium intake for a minimum period of 5 days after the serum potassium has been restored to normal is essential. Earlier cessation of potassium administration will result in failure to restore intracellular potassium and a hypopotassemia may reappear. Parenteral administration of potassium chloride is therefore continued until oral feedings can be instituted. When no more than clear liquids are tolerated, potassium salts are administered orally. A solution containing 2 grams of potassium chloride in an ounce of water is used for oral administration of potassium. One ounce of this solution taken 4 times a day provides 8 grams of potassium chloride (108 mEq. of potassium). Most foods are a good source of potassium, therefore, parenteral or oral potassium salt therapy is usually no longer needed after the patient can be fed an adequate diet.

PROPHYLACTIC USE OF POTASSIUM

Prophylactic use of potassium salts was studied in 90 patients.

Group I. Fourteen patients with a normal serum potassium received 4 grams of potassium chloride per day for 3 days (2 liters of 5 per cent glucose, each containing 2 grams of KCl) to determine the effect of intravenous potassium chloride on normal subjects. The patients were awaiting surgery for inguinal hernia, varicose veins, hemorrhoids, or leg ulcers. No toxicity was observed in any patient and the serum potassium concentration was normal 24 hours following each administration.

Group II. Seventeen patients with a normal serum potassium preoperatively received 4 grams of potassium chloride per day (2 liters of 5 per cent glucose each, containing 2 grams of KCl and 1 liter of 0.9 per cent sodium chloride solution without KCl). This was done to determine if 4 grams of potassium chloride would maintain a normal serum potassium in patients undergoing surgery for inguinal hernia, chronic cholecystitis, and uterine fibroids. Administration of potassium began 24 hours postoperatively and was continued until a liquid diet was tolerated. There was no evidence of toxicity in any patient. The serum potassium remained normal and no clinical evidence of potassium deficiency was observed.

Group III. This group consisted of 26 patients undergoing subtotal gastrectomy, hemicolectomy, or cholecystectomy and choledochostomy. Each patient received 4 grams of potassium chloride per day (administered as 2 grams of KCl per liter) beginning when parenteral fluids and Levin suction were started preoperatively and continued postoperatively until a liquid diet was tolerated. Fifteen of these patients had a normal serum potassium at all times. The other 11 patients had a normal serum potassium preoperatively and no clinical evidence of potassium deficit; however, a hypopotassemia developed by the fourth postoperative day. Each of these 11 patients lost a large volume of gastrointestinal fluids via fistulas (colostomy, cecostomy, or choledochostomy) or suction drainage.

Group IV. This group consisted of 33 patients in whom surgical procedures similar to those in group III were performed. Each patient received 4 grams of potassium chloride per day for the same period as in group III. However, 3 additional grams of potassium chloride were administered for each liter of gastrointestinal fluids removed by suction drainage or lost per fistula in a 24 hour period. A normal serum potassium was present throughout the preoperative and postoperative course of each patient.

On the basis of this experience, we believe that potassium salts should be administered prophylactically under the following conditions: (1) to patients with losses of gastrointestinal secretions by suction drainage or fistulas; (2) to patients receiving only parenteral alimentation for periods longer than 4 days; (3) to many patients undergoing extensive surgical procedures. Prophylaxis should begin when Levin suction and intravenous fluids are started before the operative day and should be reinstituted beginning 24 hours postoperatively and continued until a liquid diet is tolerated.

A solution containing 2 grams of potassium chloride (27 mEq. of potassium) in a liter of subcutaneous or intravenous fluids may be used for prophylaxis. This solution has been found to be nontoxic when administered at rates less than 120 to 180 drops (8 to 12 c.c.) per minute to patients with normal serum potassium, as long as the daily urinary output exceeds 800 cubic centimeters. Each day when any of the conditions mentioned are present the patient should receive 4 grams of potassium chloride. In addition another 3 grams of potassium chloride should be administered for each liter of gastrointestinal fluids removed by suction or lost per fistula during a 24 hour period.

SUMMARY

1. The importance of potassium deficiency in preoperative and postoperative patients is demonstrated by the fact that 314 cases were studied and treated in a general hospital during a 12 month period.

2. The pathogenic factors of potassium deficiency in surgical patients were found to be: (a) limitation of potassium intake; (b) prolonged use of potassium-free parenteral solutions; (c) loss of gastrointestinal secretions; (d) increased renal excretion of potassium re-

sulting from surgical trauma with its attendant alarm reaction, diuresis due to parenteral infusions of large amounts of sodium chloride or glucose solutions, and disturbances in acid base balance.

3. Potassium was found to be present in relatively high concentration in all types of gastrointestinal secretions. Gastric juice contained the highest concentration. The use of gastrointestinal suction drainage, without adequate replacement of potassium, was shown to lead to potassium deficit after 4 or 5 days.

4. Muscular weakness was the commonest clinical finding. Anorexia, mental depression, and abdominal distention were also common. Hypochloremic alkalosis was frequently observed and responded to administration of potassium chloride.

5. Prophylactic use of potassium salts was studied in 90 surgical patients. The indications for prophylactic use of potassium and the methods of administration are discussed.

6. Treatment of potassium deficiency is an important addition to the care of the surgical patient and is often essential for survival of the patient. Administration of potassium chloride intravenously was followed in most of the 314 cases by prompt and often striking clinical improvement.

Approximately 8,000 liters of intravenous and subcutaneous fluids containing potassium chloride were administered during this study. No complications were observed. Types of solutions and methods of administration of potassium chloride are discussed.

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HYPOTENSIVE SPINAL ANESTHESIA

NICHOLAS M. GREENE, M.D., Boston, Massachusetts

Foreword by HENRY K. BEECHER, M.D.

DELIBERATE use of the hypotensive technique in anesthesia might seem to deny several principles which many others as well as ourselves have advocated over a period of years. It seemed so to us too in the beginning, but on thinking the matter through we were obliged to admit that we did not know whether, in a man with a failing liver, for example, a long operation with a normal blood pressure was better or worse than a shorter operation with a low blood pressure. Such a question could be settled only by experience. We are in the process of obtaining that experience.

We did not lightly accept the technique, but invited Dr. John Gillies of Edinburgh to come to Boston to demonstrate it over a month's time and then sent one of our staff, Dr. Nicholas M. Greene, to Edinburgh, where, thanks to Dr. John Gillies and Sir James Learmonth, he was given every consideration and as much supervised clinical experience with this type of case as he could crowd into the 4 months that he was there.

It requires very little thought to appreciate many of the advantages, as well as the hazards, of the method. It is requiring very great effort in the laboratory to try to ferret out latent damage which possibly was caused by the method.

There is always the temptation to conclude, as long as the patient survives the ordeal, that everything has been gained and nothing lost. Only the most careful search for latent damage with negative results will justify such a conclusion. Already there have been a good many reports of serious accidents with this technique. These appear, in some instances at least, to be due to incorrect choice of the method or careless use of it. The purpose of Dr. Greene's paper is to state, as well as he can at this time, what he believes to be a careful use of the technique.

We are now engaged in studies of the effect of the method on postoperative acid-base equilibrium, electrolyte alterations, liver function, kidney clearances, and psychomotor changes. When we have a sufficient body of such data, taken in conjunction with clinical experience, it will be possible only then, to answer our original question: Does this technique make certain kinds of surgery safer than the anesthesia techniques now listed as standard?

IN 1948 Gillies (1,2,4) advocated the use of controlled hypotension produced by subarachnoid block to reduce operative blood loss. The principle of induced hypotension has since become widely accepted in Britain. Not only is Gillies' method used, but vasodepressor drugs and epidural block are employed to effect a reduction in arterial pressure. Arteriotomy is rarely used. Gillies' method has stimulated discussion in the United States, though few reports on its use have as yet appeared in the literature. After personal experience with the method at the Royal Infirmary of Edinburgh, a series of cases, to be reported later, is now under study at the Massachusetts General Hospital. This potentially valuable method of anesthesia entails certain risks; its indiscriminate or inexperienced use will greatly increase this risk. Therefore, the purpose of this paper is to outline the technique, to review its advantages and dangers, and to indicate its possible sphere of usefulness.

The goal of hypotensive spinal technique is to diminish bleeding in the surgical field. This is accomplished by reducing the arterial pressure with an extensive subarachnoid block. The advantages of such hypotension are: (1)

From the Department of Anesthesia of the Massachusetts General Hospital, Boston.

increased technical freedom and speed in certain operations, and, in fact, some procedures are made surgically feasible that would not otherwise be possible;[1] (2) reduction in duration of anesthesia and operation; (3) associated maximal relaxation in abdominal cases; and (4) reduction of the number of transfusions necessary.

Because of the different concentrations of a local anesthetic agent required to block various types of nerve fibers, a complete sympathetic block may be attained, while sensory anesthesia is less extensive, and motor paralysis even more limited. Thus sympathetic fibers at the first thoracic level may be safely blocked by a dilute solution while the phrenic and upper 3 or 4 intercostal nerves continue to function. The term "total spinal" is inaccurate when referring to this type of anesthesia, and should refer only to a total sensory block as done by Koster, Jonnesco, and Wright. "Hypotensive spinal" or "the Gillies technique" are more precise terms.

METHOD

Although the minutiae of the technique are best illustrated by a presentation of cases (3) with extensive studies of their metabolic effects, certain basic procedures should be emphasized. Before the spinal injection the patient must be put to sleep. It is important that the distressing subjective symptoms of hypotension be obviated. In addition, it is better to render the patient unconscious beforehand, so that the effects on the cardiovascular system of the general anesthetic agent alone can be evaluated. The administration of a general anesthesia to a conscious patient already hypotensive can be dangerous. Thiopental in single large doses of 500 to 800 milligrams is used to induce anesthesia, and a completely patent airway is then assured. Endotracheal intubation should be performed if indicated, with small doses of curare if necessary, though the thiopental combined with skill, adroitness, and a sense of correct timing is often sufficient.

[1]Surgery had been attempted on a vascular cerebellar tumor on 2 occasions but had to be abandoned both times because of great bleeding. A third attempt with this technique was successful.

The patient is unconscious when the spinal tap is being performed and during the hypotension this is accomplished by nitrous oxide oxygen. Any initial "bucking" on the endotracheal tube may be stopped by the temporary addition of small amounts of cyclopropane. While the blood pressure is down the patient is kept asleep with nitrous oxide oxygen in a ratio of one to one. This will usually also provide sufficient anesthesia, though without relaxation, for operations performed outside the area made anesthetic by the spinal block. If more general anesthesia is needed, the nitrous oxide should never be increased over a proportion of two to one. Instead, if needed, cyclopropane in low concentrations may be used. Further thiopental should not be given to such a patient.

After the patient is asleep and an airway established, he is put into the lateral position for the spinal puncture. Clinically an adequate sympathetic block is obtained by injecting one of the usual hyperbaric local anesthetic agents into the lumbar subarachnoid space with the patient in the Trendelenburg position. The routine use of hypobaric solutions is not advocated, since the head should be down while the patient is made hypotensive. The dosage used should be about one-quarter again as much as if a routine high abdominal block were to be obtained. Procaine (procaine hydrochloride U.S.P.), pontocaine (tetracaine hydrochloride U.S.P.), or nupercaine (dibucaine hydrochloride N.F.) is used, depending on the expected duration of the surgery. For cases lasting longer than 3 hours, a subarachnoid catheter may be inserted and fractional doses of the local anesthetic agent given. The concentration of the agent in its diluent should be the same as that used to obtain the usual spinal anesthesia. Increasing the speed of injection and barbotage are not advised. The necessary height of the block can be obtained by using the Trendelenburg position of about 15 degrees. The extent of the sympathetic block is regarded as satisfactory when the systolic pressure falls to about 65 to 85 millimeters of mercury. The patient may then be placed in position for surgery. This alteration of position usually produces a further drop in systolic pressure of from 15 to

20 millimeters of mercury. The hypotension is regarded as adequate if the systolic pressure is between 40 to 70 millimeters of mercury during the surgery. Levels below this are inadvisable, while higher ones are ineffective in reducing bleeding. One can often ascertain whether a complete sympathetic block has been obtained by observing whether a bradycardia is present, by the appearance of bilateral Horner's syndromes, and by the absence of sudomotor and pilomotor activity on the face and neck. It is impossible to determine the sensory level of the block. The motor level can be estimated by the appearance of intercostal paralysis.

The position of the patient during surgery is of great importance. The operative area must be uppermost in order to avoid troublesome and dangerous venous oozing. The head must also be down in order to assure adequate cerebral circulation. The proper position for pelvic cases, for example, would be moderate Trendelenburg position, the pelvis at the break of the table, with the lower extremities slightly extended at the hips by dropping the distal end of the table. Alteration of the position so as to increase or decrease venous return to the heart, while keeping the operative area elevated, has also proved to be an effective means of controlling the degree of hypotension under these conditions.

The normal reactions to blood loss, hypoxia, or hypercapnia depend to a large extent on the autonomic nervous system. When there is an extensive sympathetic block the usual responses to such conditions are obtunded or absent. Patients made hypotensive by this method must, therefore, be very closely watched. Their color, pulse, and blood pressure must be continuously observed. The capillary filling time is a useful guide and should be frequently tested. The best indication of the patient's condition is the character and rhythmicity of the respiration. A change from the slow, deep regular respiratory motions usually present in these cases to irregular, jerky movements may be the first indication of serious deterioration of the patient's condition. This change is probably evidence of inadequate medullary blood flow. Because of the importance of spontaneous respirations, controlled or even assisted respirations should be avoided, unless the respirations become inadequate. This type of anesthesia is, therefore, unsatisfactory when the pleural cavity is open. In addition, positive intrapulmonary pressure may prove deleterious to the cardiovascular system in such cases.

The blood pressure should be raised when any of the following occur: (1) alteration in character or rhythmicity of respirations; (2) prolongation of the capillary filling time; (3) cyanosis in the presence of a patent airway and an adequate concentration of oxygen in the inspired air; (4) sudden drops in blood pressure after it has been stabilized; (5) alterations in rate or rhythm of the pulse; and (6) unmeasurable blood pressure, even with a palpable pulse, at establishment of hypotension.

There are two methods by which the pressure may be raised: alteration of position and the use of vasopressors. Changing the position so as to increase venous return to the heart usually raises the pressure about 20 millimeters of mercury within several minutes under these conditions. This is accomplished by elevation of the lower extremities. Further exaggeration of the head down position is not helpful. If the situation is more urgent, a vasopressor (already drawn up) should be given intravenously. The desirable agent acts rapidly, exerts a prolonged action, and produces its effect primarily by causing peripheral vasoconstriction rather than by increasing cardiac output. Desoxyephedrine (methamphetamine hydrochloride U.S.P.) and vasoxyl (methoxamine hydrochloride) have proved satisfactory in intravenous doses of 10 and 7.5 milligrams respectively.

During surgery the little bleeding that does occur must be carefully controlled. If there is meticulous hemostasis there is no increased incidence of postoperative hematomas, probably because of the formation of intravascular clots in the smaller vessels during the hypotensive period. Large vessels which do not form such clots will bleed even during the period of reduced arterial pressure and so should be clamped. Because spinal anesthesia reduces the ability to withstand hemorrhage, any significant blood loss (over 250 to 300 c.c.) should be replaced immediately.

As the operation nears completion the pressure may be raised, although usually this is not necessary, for the dose of the anesthetic agent can be gauged so that the pressure rises spontaneously at that time. The patient should be under close supervision in the Trendelenburg position postoperatively until there is a return of sympathetic tone.

Postoperatively these patients react rapidly and experience little or no nausea or vomiting. To date there has been no clinical evidence of an increased incidence of cerebral, myocardial, renal, or hepatic complications. These matters are now under investigation.

Selection of the patient. Absolute contraindications to the use of this type of anesthesia are the following: (1) coronary artery disease or insufficiency, (2) generalized arteriosclerosis, (3) aortitis, (4) aortic valvular disease, (5) congenital heart disease, (6) anemia, (7) reduced blood volume, (8) impending or actual peripheral vascular collapse, (9) polycythemia, (10) interference with normal oxygenation, (11) decreased cerebral blood flow, and (12) conditions contraindicating spinal or thiopental anesthesia.

While it is probable that under this type of hypotension the coronary blood flow is adequate for the oxygen requirements of the normal myocardium, the same cannot be assumed to hold in the presence of abnormal coronary arteries.

Congenital heart disease, with the exception of coarctation of the aorta, contraindicates this technique, for in the presence of abnormal shunts systemic hypotension may further impede arterial oxygenation.

Anemia is another contraindication when the hemoglobin is reduced to 80 per cent of normal or below. To assure adequate tissue oxygenation with this method there must be a normal amount of hemoglobin, fully saturated. Similarly, a significant reduction in blood volume makes this method inadvisable. Patients in or bordering on peripheral vascular collapse from trauma, toxins, or sepsis, even though with a normal hemoglobin and blood volume, should not be given this type of anesthesia. Polycythemia is a contraindication because of the concomitant tendency to thrombus formation.

Pathologic conditions grossly interfering with diffusion of gases across the alveolar membranes also contraindicate this type of anesthesia (e.g., pneumonia, pleural effusions, asthma, emphysema, pulmonary fibrosis, or a high, fixed diaphragm). Less marked pulmonary pathological states (atelectatic patches or small pleural effusions) are regarded as relative contraindications.

Patients exhibiting or suspected of having a below normal cerebral blood flow should not be made hypotensive, since reduction of systemic pressure may further reduce cerebral blood flow to the point where brain tissue will be seriously damaged.

Age alone is not a contraindication, though above the age of 65 years there is generally enough concurrent disease to make this method inadvisable.

Selection of the operation. Operations in which this technique is to be used must be major enough to warrant the increase in risk involved in the use of this method over the usual techniques. The surgery must normally entail bleeding which is troublesome, dangerous, and which may jeopardize the success of the operation. The operation, furthermore, must be one in which the requirements mentioned as to position can be met to the satisfaction of both surgeon and anesthetist.

Selection of surgeon and anesthetist. The surgeon operating with this type of anesthesia must understand its dangers and its limitations, especially in regard to position. He should be gentle, experienced, and fast since it is not desirable to maintain the hypotension longer than necessary. The anesthetist must fully comprehend all of the physiological aspects of the technique, be alert at all times, and be capable of thinking and acting rapidly under unusual circumstances. He should have wide experience with spinal anesthesia and preferably should observe this particular technique several times before attempting it himself. He must have the complete confidence of the operating surgeon.

DISCUSSION

The physiological implications of this type of anesthesia are interesting. Unfortunately they are complex and cannot be adequately

discussed here. Many of these problems, especially those relating to renal, hepatic, and cellular physiology are under active study in the present series of cases (3), together with supplementary animal experiments. Suffice it to say that observation of our own cases, plus a review of the literature too extensive for discussion, has led to our belief that when properly managed this method of anesthesia in carefully selected cases is physiologically reasonable for trial.

The use of intravenous autonomic blocking agents has been advocated for the production of similar hypotension. Despite the greater ease of an intravenous injection compared with lumbar puncture, especially if a catheter is to be inserted into the subarachnoid space, the spinal technique so far seems preferable to us. The spinal block provides not only hypotension but also anesthesia and relaxation, while hexamethonium, for example, provides only autonomic block. The effects of hexamethonium on the blood pressure are unpredictable: a severe hypotension may develop after 10 milligrams in a patient, while in another, 100 milligrams may not have an adequate effect. This degree of uncertainty is not present with spinal block. Often the only way to obtain adequate hypotension with hexamethonium is by using a reverse Trendelenburg position. It does not appear sound to have the head elevated at such a time. And, finally, the methonium compounds are new and have undesirable side effects. On the basis of experience with both hexamethonium and the spinal technique, we favor the latter at present.

SUMMARY

A brief description of the clinical aspects of the hypotensive spinal technique is presented, together with its contraindications and areas of usefulness. If properly employed this method of anesthesia appears to be of real benefit to the surgeon and patient alike. The method deserves conservative trial and further investigation.

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THE SURGICAL TREATMENT OF VARICOSE VEINS IN PREGNANCY

FRANK W. QUATTLEBAUM, M D., F.A.C.S., and JANE E. HODGSON, M.D.,
St. Paul, Minnesota

IT has been only in the past few years that active therapy of varicose veins during pregnancy has been advocated by even a few. Even local injections with sclerosing solutions were considered radical therapy in the past. McPheeters (5) formerly advocated ligations for the severe cases in pregnancy, but later abandoned this procedure in 1945 as "100 per cent unsuccessful." The only therapy Dr. McPheeters (6) is advocating at the present time for the pregnant woman with varicose veins is hormonal.

A few articles on the therapy of varicose veins in pregnancy have appeared in the literature in the last 2 to 3 years. The majority of these concern the use of injections and/or ligations, and the more radical stripping procedure is reserved for the most severe cases only. Hugh Hamilton, one of the few advocates of radical therapy of varicose veins during pregnancy, states that they are "doing fewer and fewer cases by ligation and injection or injection alone, and are leaning more to ligation and stripping." Sullivan from the University of Wisconsin recently reported a series of ligations in 34 pregnant women. The author mentions that stripping of the entire saphenous system was used in the very severe conditions, but apparently not as a routine procedure. Peyton and Loop, in 1949, reported 20 cases of saphenous phlebectomies in pregnant women. Shank, in 1948, reported a group of 30 cases of pregnant women in which high ligations combined with segmental resection of the saphenous system were performed. To our knowledge, there has been no published series in which saphenous ligation and stripping were performed during pregnancy on all patients in whom radical therapy was indicated.

ETIOLOGY

There are many etiological factors that play a role in the production of varicose veins. The pregnant state is merely an additional one. Varicose veins are a constitutional defect, such as graying of the hair and wrinkles. Other existing situations, no doubt, augment or speed up the process. The pregnant state is one of these. Anatomical variations, defective valves, occupation, frequent pregnancies at short intervals are some of the factors interfering with the recovery of veins that have been dilated during pregnancy. A competent and strong saphenous system may become relatively incompetent and dilated during pregnancy because of its normal elasticity, but many of these veins recover completely. Failure to recover, perhaps due to one or several of the factors mentioned, results in permanent varicosities and varying degrees of incompetency.

That the pressure factor is important during pregnancy cannot be denied. Pascal's law applies; namely, that pressure exerted upon an enclosed fluid is transmitted undiminished in all directions and acts with equal intensity on all surfaces. The sump action of the right heart mitigates Pascal's law, except when the patient is straining or coughing. At that time, the sump effect is nonexistent. In 1939, Adams made an important contribution toward the complete understanding of the cause and surgical treatment of varicose veins by differentiating between pure gravity effect and that of voluntary muscular action such as coughing and straining. He found, as has been done before him, that the standing pressure in the saphenous system before and after high ligations is identical. However, after interruption, the significant alteration is a decrease in straining pressure. It is to be remembered that hydrostatic pressure is transmitted through the communicating veins between the superficial and deep systems, unless the two systems are disconnected by interruption at the affected levels.

Veal, in 1941, measured the popliteal vein pressure while the patient was in the standing position and while she was doing specified exercises. This was done in a group of pregnant women, some having varicose veins and edema, and the remainder having no difficulty regarding the lower extremities. Consistently, his pressure readings showed a more severe type of venous obstruction in the ileofemoral tree in those women who had varicose veins and edema. Those with no varicose veins or edema had a pattern of a less severely obstructed flow. It has long been recognized that there is an increased femoral vein pressure as pregnancy progresses, whether the pressure readings are taken in the upright or supine position. Further evidence, which Veal points out, to support the obstructive origin of edema in varicose veins in pregnancy, is the fact that there are occasional patients who have a very severe degree of unilateral varicose veins. A high popliteal vein pressure exists on one side, whereas the normal side has a relatively normal pregnancy pressure reading.

INCIDENCE OF VENOUS DISEASE IN PREGNANCY

Our recent analysis of the case histories of 600 consecutive pregnant women in private practice reveals the incidence of varicosities to be 66 cases, or 11 per cent. Nineteen of these women were observed to have a definite episode of thrombophlebitis, either antepartum or postpartum, and 3 gave histories of thrombophlebitis with previous pregnancies. This total of 22 women with thrombophlebitis resulted in a rather startling incidence of 3.6 per cent of the entire group, or 33.3 per cent of those patients with varicosities (Tables I and II). This high incidence of thrombophlebitis in our series was an incidental and unexpected finding and one that may be questioned by fellow obstetricians who claim a much lower incidence of venous disease in pregnancy. Admittedly many in our series were cases of mild and superficial phlebitis frequently overlooked or ignored by clinicians. However, inasmuch as there is no definite demarcation between involvement and subsequent damage of the superficial and deep venous systems, we believe that the so-called mild cases are significant forewarnings of future vascular damage and should, therefore, be treated with the greatest of respect by the clinician. The aggravation of labor and delivery may later produce the classical picture of thrombophlebitis or phlebothrombosis. There were 2 cases of nonfatal pulmonary embolus—1 occurring 8 hours postpartum, the other on the sixth postpartum day. Many of the cases of phlebitis originate in the antepartum period, are mild in character, and frequently overlooked by the clinician. It is therefore evident that the treatment of varicosities in pregnant women represents a real problem—not only from the standpoint of the patient's comfort and cosmetic effects, but also in regard to the prophylaxis of thromboembolic phenomena. It is now generally agreed that active therapy of varicose veins tends to reduce the incidence of subsequent thromboembolic disease in surgical patients, and there is no reason why this same fact does not obtain in obstetrics. None of our cases of thrombophlebitis coexisted with any postpartum pelvic infection; and we do not believe that the element of postpartum sepsis played any causative role.

With the recent reduction of maternal deaths from the triad of hemorrhage, infection, and toxemia, deaths due to pulmonary emboli are assuming a greater relative importance in maternal mortality surveys. In

TABLE I.—STUDY OF VARICOSE VEINS IN 600 CONSECUTIVE PREGNANT WOMEN

	No. of cases	Per cent of total
Incidence of varicose veins	66	11.0
Incidence of phlebitis	22	3.6
Incidence of pulmonary embolism (nonfatal)	2	.33

TABLE II.—ANALYSIS OF 66 PREGNANT WOMEN WITH VARICOSE VEINS

	No. of cases	Per cent of total
Incidence thrombophlebitis	22	33.0
Incidence pulmonary embolism (nonfatal)	2	
Patients receiving conservative therapy	52	78
Patients receiving radical therapy	14	22

TABLE III.—COMPARISON OF RADICAL AND CONSERVATIVE THERAPY

	Radical treatment	Conservative treatment
Total No. of patients	14	52
Incidence of subsequent thrombophlebitis	1	21
Incidence of pulmonary embolism (non-fatal)	0	2

a recent analysis of 47 maternal deaths in the rural south (3), there were 7 deaths from pulmonary emboli. As a cause of maternal mortality, pulmonary embolus is surpassing infection in relative frequency, being the cause of death in 10 to 20 per cent of cases. Apart from the foregoing, an additional important argument for the active therapy of varicose veins in pregnancy is that the women in this group, more than in any other, have numerous demands upon them at home and at work, which makes attempts at conservative therapy almost an impossibility. For some of these women, being incapacitated even for a relatively short time involves great economic and social hardship. The two chief arguments used against the active therapy of varicose veins in pregnancy are in themselves directly contradictory: namely, (1) treatment is useless in pregnancy because the varicosities disappear or improve after pregnancy has terminated; (2) treatment is useless because varicosities recur as fast as they can be eradicated. In regard to the first argument, it is obvious that varicosities that disappear or improve after pregnancy are mild cases which do not require radical therapy. In regard to the second objection, varicosities will not recur as fast as they can be eradicated if a radical enough stripping procedure is performed.

TABLE IV.—INDICATIONS FOR SURGERY

	Severe varicose veins No. cases	Incapacitating varicose veins No. cases	Total No. of cases
No. of patients	18	5	23
Interval varicose veins	15	3	18
History of phlebitis	9	1	10
Stasis ulcers	2	0	2
Vulvar varicosities	0	2	2
Previous ligation	5	1	6

INDICATIONS FOR SURGERY

In our group of 66 women with varicose veins, ace bandages and injections of sylnasol sufficed in approximately 75 per cent. However, it was interesting to note that it was in this conservatively treated group that all but 1 of the cases of antepartum and postpartum phlebitis occurred (Table III). In the remaining 25 per cent, which represented 17 cases, radical vein stripping was advised. Indications for radical surgery were: (1) incompetent varicose veins existing between pregnancies; (2) previous thrombophlebitis; (3) vulvar varicosities, which were incapacitating in character. In our experience, it is impossible to apply enough pressure on the vulvar areas to satisfactorily relieve the patient of her symptoms. Of the 17 patients advised to have radical surgery, 14 were operated upon, regardless of the period of gestation. The old bugaboo, which deprives a pregnant woman of necessary surgery, because of the fear of initiating premature labor, has been fairly well eliminated. In none of our cases was the pregnancy jeopardized in any way by the radical surgery. In addition to our 14 patients receiving radical surgery, 9 patients were referred by other obstetricians. These 23 individuals received saphenofemoral ligation and stripping of the greater saphenous vein, plus eradication of any other incompetence of the superficial system that was indicated. These 23 cases have been divided into 2 groups, as far as indications for surgery are concerned (Table IV).

SURGERY AND RESULTS

One-fourth of these patients were operated upon during the first trimester; three-fourths had been done by midterm. Six were operated upon during the third trimester, all 6 of whom were physically incapacitated—2 by vulvar varicosities, and 4 by leg varicosities.

In 12 of the 23 patients a unilateral procedure was done; in 11 a bilateral procedure. In addition, in 5 instances operations on the lesser saphenae were done, and in 3 complete excision of vulvar varicosities. Individual excisions of so-called "blow-outs," or incompetent perforators, were done in all cases, whenever indicated. It is interesting that over half

these procedures were indicated in patients with unilateral varicosities, which contradicts those proponents of the theory that varicosities are chiefly influenced by hormonal variations in the pregnant state. Most of these unilateral varicosities persisted during and after a previous pregnancy.

After surgery, an average of 2.4 postoperative injections and .6 postpartum injection were required to clear the leg completely of any terminal varicosities. It has also been noted that fewer postoperative injections are required for the nonpregnant patient when stripping is used instead of a less radical procedure.

Although varicose veins can be stripped under local anesthesia, sodium pentothal was used in these 23 cases. The procedure consists of a routine saphenofemoral ligation, in which all the criteria for a proper ligation are observed. In addition, the terminal trunk of the greater saphenous vein is divided just medial to the medial malleolus and stripped in the retrograde manner from the ankle to the groin. Should the lesser saphenous be incompetent, it is likewise ligated and stripped. Any large branches of the greater saphenous that can be excised or stripped are so treated. In addition, any incompetent perforators or so-called "blow-outs" are excised. A slight Trendelenburg position is used, inasmuch as the lowering of femoral vein pressure mitigates excessive bleeding. Although innumerable strippers are available, we use the Myers intraluminal stripper, devised by Dr. Tom Myers of Rochester, Minnesota. In addition, a Babcock intraluminal and a Mayo extraluminal stripper are included in the sterile setup and are used in certain situations.

Heavy pressure dressings are applied for 24 hours by using two folded towels over the medial side of the leg from ankle to groin encompassed by two 4 inch ace bandages from the ankle to the upper thigh. The patient is encouraged to walk on the evening of surgery, and is allowed to be up as she desires. The pressure dressings are removed in 24 hours, and the patient is instructed to use an ace bandage from the ankle to the knee until the sutures are removed and the postoperative injections are completed.

The patient is allowed to go home in 24 to 48 hours. It was noted in the beginning that when the stripping procedure was used in the pregnant patients, they seemed to recover more quickly than the nonpregnant women. They apparently are less incapacitated and are anxious to go home. This fact is perhaps affected by the youth of the patients in this group, as well as the many responsibilities that they have at home with their young families. It has been noted with all vein stripping that postoperative discomfort is surprisingly mild and definitely less than that following a routine saphenofemoral ligation and retrograde injection.

The patient returns to the office in 1 week; the sutures are removed; and injections are begun for those small terminal twigs not eliminated at the time of surgery. The surgeon does not dismiss the patient until after her delivery. Although one can completely eradicate all terminal veins by postoperative injections, one must still watch these patients until delivery, as they are prone to develop further small dilatations. These can be cared for by simple injections; and after delivery, the patient is seen by the surgeon for a final time when she returns to the obstetrician for her 6 weeks postpartum examination. At that time, if any residual varicosities are present, they are injected; and the patient is then instructed to return to the office at periodic intervals for from 6 months to 1 year.

Another routine followed with these patients is the use of ace bandages while the patient is in labor and in the stirrups on the delivery table. Trauma to varicose veins undoubtedly plays a role in the production of phlebitis, and we have felt that some protection to the veins is afforded by the routine use of bandages.

Admittedly, the treatment of varicose veins during the pregnant state is harder and requires more time than in the nonpregnant state. Successful results in varicose vein surgery in any situation depend chiefly upon the quantitative removal of veins. The pregnant state allows the operator some advantage from this viewpoint. With the distention of the main trunks, together with their large branches, and particularly with the prom-

inence of the perforators that have become incompetent, one is more likely to eliminate all of the veins that are possibly causing trouble, or that might remain prominent after pregnancy has terminated.

In analyzing the results obtained in this group of pregnant women, the patients were contacted currently, which is from 2 months to 2½ years following their surgery. They were likewise followed during their pregnancy by both the obstetrician and peripheral vascular surgeon. The results obtained during pregnancy and after delivery, as far as the patient was concerned subjectively and as far as the physicians were concerned objectively, were excellent in all cases.

CONCLUSIONS

1. It is our impression that the ligation method of varicose vein treatment in general is an inadequate operation. We are convinced that a thorough ligation and stripping procedure is more satisfactory, both in the pregnant and nonpregnant state. The fact that, of 23 pregnant patients undergoing surgery, previous ligations with exceedingly poor results had been performed in 6 illustrates this point.

2. Admittedly vein stripping is more difficult in the pregnant patient from a technical point of view; however, that in itself is insufficient reason for delaying treatment until pregnancy is terminated.

3. These patients are most grateful for the relief afforded them by the surgery. It would seem that the treatment of symptomatic varicose veins during pregnancy can be carried out as safely and with as permanent results as in the nonpregnant state.

4. From an obstetrician's point of view, the treatment of varicose veins in pregnancy by radical stripping is extremely satisfactory. Five patients have been observed through subsequent pregnancies, during which they have been able to discard their ace bandages. As we observe more of these patients who have had strippings, we are becoming less conservative and advising a larger percentage to have surgical treatment.

5. Obstetricians should be continually alerted for the signs of mild phlebitis appearing during the antepartum and postpartum periods. We believe the incidence of venous disease in pregnancy is higher than previous literature would indicate.

6. Pregnancy is no contraindication to radical surgery when such treatment of varicose veins is indicated. The pregnant state with its aggravation of venous disease actually is additional reason for giving the patient prompt and adequate treatment which will not restrict her activities.

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LOCAL RECURRENCE FOLLOWING SUBTOTAL RESECTION FOR GASTRIC CARCINOMA

FRANK B. THOMSON, M.D., and R. EDWARD ROBINS, M.D.,
Vancouver, British Columbia

THE 5 year survival rate following subtotal gastrectomy for carcinoma of the stomach is reported to be approximately 20 per cent when radical local excision is carried out. In average surgical practice it is probable that only those patients with carcinoma in situ or low grade tumors, who might have survived 5 years anyway, are salvaged by the usual subtotal gastrectomy.

Why does subtotal gastrectomy fail? Is it because distant metastases, microscopic in size, have already occurred at the time of operation? While this may well be the case, to accept this thesis would mean that palliative resection should be the rule. In the absence of conclusive proof that this is so, surgeons are unwilling to adopt such a pessimistic attitude. On the assumption, therefore, that in a reasonable number of cases, distant metastases of a microscopic size have not occurred at the time of resection, a study of the incidence of local recurrence in gastric or duodenal stumps and immediate lymphatic drainage should help to reveal some of the shortcomings of this operative procedure.

From the Vancouver General and Shaughnessy Hospitals, we have obtained the records of 28 patients, each of whom, after a previous subtotal gastrectomy for carcinoma of the stomach, had a postmortem examination. Following a study of these records, local recurrences in gastric stump, duodenal stump, and gastric bed were tabulated as follows:

	Gastric stump No. cases	Duodenal stump No. cases	Gastric bed No. cases
Recurrence	5	3	13
Negative	17	25	9
Questionable	6	0	6
Total	28	28	28

From the Department of Surgery, University of British Columbia School of Medicine.

By the term gastric bed is meant the immediate perigastric tissues including the whole of the pancreas. In 6 cases, because of massive local recurrence, it was impossible to be sure whether the recurrence originated in gastric stump or gastric bed. These have been listed as questionable.

In these cases, the incidence of gastric stump recurrence is approximately one-half that in the gastric bed. In a recent study of a similar nature, which was carried out by McNeer and his associates, they reported their findings as follows:

	Gastric stump (all cases)	Duodenal stump (alone)	Gastric bed (alone)
Recurrence	46	9	19
Negative	46	78	44
Total	92	87	63

These figures seem to indicate a much higher relative incidence of gastric stump as compared to gastric bed recurrence. However, in reporting all cases of gastric stump recurrence and only those cases of gastric bed recurrence in which the gastric stump was negative, they have assumed that in those cases in which both gastric stump and gastric bed are involved, the recurrence in the gastric bed arose subsequent to the gastrectomy and also as a secondary spread from the recurrence in the gastric stump. It is at least as probable that microscopic gastric bed secondaries were already present at the time of operation in these cases and to assume that they arose postoperatively is not justified. Also, if in these cases the gastric bed secondaries all arose as spread from the gastric stump recurrence, then a study of those cases with gastric stump recurrence should reveal a higher incidence of gastric bed recurrence than in those cases with a negative gastric stump. In this regard, a study of McNeer's figures reveals the following:

	Total No. of cases	Gastric bed recurrence No. cases	Per cent
Gastric or duodenal stump recurrence....	54	29	54
Gastric or duodenal stump negative.....	38	19	50

In our own smaller series, the findings were as follows:

	Total No. of cases	Gastric bed recurrence No. cases	Per cent
Gastric or duodenal stump recurrence....	7	4	59
Gastric or duodenal stump negative.....	17	11	65

TABLE I.—SITE OF LOCAL RECURRENCE

	Gastric stump		Duodenal stump		Gastric bed		Local recurrence present
	No.	Per cent	No.	Per cent	No.	Per cent	Per cent
McNeer and Associates 92 cases	46	50	14	15	48	53	80.5
Vancouver General Hospital and Shaughnessy Hospital 28 cases	5–11	18–40	3	11	13–19	46–68	93

The percentages in both series are so close as to suggest that the gastric bed recurrence rate is independent of the presence or absence of gastric or duodenal stump involvement, and therefore that microscopic secondaries in the gastric bed are already present at the time of operation in the same percentage of patients subsequently developing gastric stump recurrence, as in those who do not. Therefore, all cases of gastric bed or duodenal stump recurrence should be included and not just those in which the gastric stump was negative. With this conclusion in mind, it is possible to present the incidence of gastric or duodenal stump and gastric bed recurrence in McNeer's series and to compare this with the smaller present series (Table I).

It remains, therefore, to consider the incidence of local recurrence as tabulated, and from this information, attempt to determine why subtotal gastrectomy fails to prevent local recurrence of gastric carcinoma.

Gastric stump recurrence is sufficiently frequent to suggest that resections are too often done through involved gastric tissue. McNeer and his associates (5) report gastric recurrence in 50 per cent of cases. The exact incidence in our series is confused by the presence of 6 cases in which it was not possible to be sure where the local recurrence originated. That a similar difficulty is not reported in McNeer's series is surprising. The classification of all such doubtful cases as gastric stump recurrence would, of course, markedly raise the apparent incidence of this complication in any series (from 18 to 40 per cent in the present series). Coller and Kay found carcinoma to the line of gastric section in 24.5 per cent of surgical specimens examined. Zinninger and Collins reported a similar finding in approximately 15 per cent of surgical specimens. Lahey and Marshall quote a personal communication from Shields Warren who found gastric stump recurrence in 19.6 per cent of 122 patients coming to post mortem having had a previous subtotal gastrectomy for carcinoma. It would seem from these reports and our own experience, that the line of gastric section is too low in roughly 30 per cent of cases. Does this mean that total gastrectomy should be the rule for all carcinoma of the stomach? This has been advocated in recent reports by McNeer (5) and Lahey. Wangensteen (6) in 1947 stated that it remained to be proved that total gastrectomy would materially improve the 5 year survival rate. Longmire in 1947 stated that the postoperative disability after total gastrectomy was such that it should not be a routine procedure for carcinoma of the stomach. Walters and his coworkers in 1947 were not in favor of routine total gastrectomy unless it was obviously necessary reasonably to expect to remove all the cancerous tissue in the stomach. In their opinion, adequate local lymph gland removal is just as possible with subtotal as with total gastrectomy. While it may be too soon to judge, there has been no series in the surgical literature to date showing any significant improvement in the cure rate by routine total gastrectomy. The functional result is so much better after subtotal resection that a portion of the stomach should be left if at all possible. West, in discussing a series of total and subtotal resections, noted a much better nutritional status in the subtotal as compared to the total resections. How then

are we to prevent the 30 per cent gastric stump recurrence and still avoid the functional disability of routine total gastrectomy? Seventy per cent of gastric carcinoma occur in the prepyloric area and 30 per cent in the body or cardia. Certainly subtotal resection should be adequate in those prepyloric tumors that show no gross evidence of extensive proximal intramural infiltration. Zinninger and Collins, from their study of surgical specimens, stated that the line of gastric section should be 2 inches above what one would consider to be safe and beyond all gross evidence of infiltration. McNeer and his associates (4) take a circular quick section at the line of gastric division. Subtotal gastrectomy should be an adequate procedure in a significant number of cases if the line of gastric section were kept at least 2 inches proximal to all gross evidence of intramural infiltration in those cases of prepyloric carcinoma without extensive proximal infiltration. It should be noted that while total gastrectomy may be indicated in the widely infiltrated cases, the incidence of distant metastases at the time of operation in such cases is so high that total gastrectomy should nearly always be considered a palliative procedure.

The duodenal stump is also an important site of local recurrence. That pyloric carcinoma directly invades the first portion of the duodenum was clearly demonstrated when Coller and Kay found duodenal involvement in 26 per cent of surgical specimens. Zinninger and Collins in a similar study reported 25 per cent duodenal spread. McNeer (5) found recurrence in the duodenal stump in 15 per cent of post mortems after partial gastrectomy. In our smaller series there were 3 cases (11 per cent) of duodenal stump recurrence. Certainly the whole of the first portion of the duodenum must be resected in any partial gastrectomy for carcinoma of the pyloric end of the stomach if duodenal stump recurrence is to be prevented.

Local recurrence in the gastric bed is even more frequent than in gastric or duodenal stumps. Consideration of our own small series and McNeer's larger one suggests that slightly over 50 per cent of cases recur in the local lymphatic drainage area. Also it seems likely, as mentioned previously, that this area is already microscopically involved at the time of resection and not secondary to recurrence in gastric or duodenal stumps. The anatomical distribution and the frequency of recurrence in the various components of this lymphatic drainage were investigated by Coller and Kay. They described the group of glands lying over the head of the pancreas and adjacent to the distal portion of the greater curvature and first and second portions of the duodenum as the inferior gastric group. This group drain the pylorus and the first portion of the duodenum, and they were more frequently involved than any others (65 per cent of cleared surgical specimens). Of those specimens with positive inferior gastric glands, the carcinoma extended to the line of section in this area in over 60 per cent. Therefore, because the greater part of this group are technically inseparable from the head of pancreas and duodenum, residual carcinoma must have been left in this area in over 60 per cent of their cases. They performed 3 autopsies after partial resection and in 2 cases found involved glands about the head of the pancreas and the duodenal stump. Coller and Kay state that "residual carcinoma is frequently left in this area." The glands in the lesser omentum and along the course of the left gastric artery known as the superior gastric glands were involved in over 40 per cent of their cases. These drain to the celiac glands lying at the superior border of the pancreas at the junction of body and tail. Of the cases in which this group was involved, there was carcinoma to the line of section and therefore residual carcinoma left in this area in 65 per cent. The suprapyloric glands about the hepatic artery also drain into the celiac group and were positive in over 20 per cent of specimens. The pancreaticolienal glands along the proximal portion of the greater curvature and the superior border of the tail and body of the pancreas were involved in over 13 per cent of specimens.

It should be evident from the studies of Coller and Kay and from a knowledge of lymphatic anatomy that the lymphatic drainage of the stomach lies to a considerable extent in the peripancreatic tissues, and particularly about the head of the pancreas. A major por-

tion of the infrapyloric glands and almost all the celiac and pancreaticolienal glands are intimately associated with the pancreas. This major portion of the immediate lymphatic drainage of the stomach, lying as it does in the peripancreatic tissues and technically inseparable from the pancreas itself, is not removed during radical partial gastrectomy for carcinoma of the stomach as it is carried out at present. Because of this, the highest incidence of local recurrence (over 50 per cent) is found in this area. It would appear from this study that local recurrence in the immediate lymphatic drainage area of the stomach is extremely frequent and that from a pathological standpoint it could only be significantly reduced by the addition of total pancreaticoduodenectomy to the radical operative treatment of selected cases of carcinoma of the stomach. Whether such a radical approach is feasible could only be determined by experience.

CONCLUSIONS

1. In those patients with carcinoma of the stomach who develop recurrence in the immediate lymphatic drainage area (gastric bed) after partial gastrectomy, it is more likely that these glands were already the site of microscopic secondaries at the time of operation, than that they became involved because of secondary spread from subsequent recurrence in the gastric or duodenal stumps.

2. The gastric stump recurrence rate following partial gastrectomy for carcinoma of the stomach is approximately 30 per cent.

3. The duodenal stump recurrence rate following partial gastrectomy for carcinoma of the stomach is 10 to 15 per cent. This would be significantly reduced if the whole of the first portion of the duodenum were routinely resected in all operations for carcinoma of the stomach.

4. A significant portion of the immediate lymphatic drainage of the stomach lies in the peripancreatic tissues and is technically inseparable from the pancreas.

5. This commonly involved portion of the immediate lymphatic drainage is not removed in radical partial gastrectomy as at present carried out for gastric carcinoma. For this reason there is local recurrence in this area in over 50 per cent of cases.

6. This peripancreatic portion of the immediate lymphatic drainage of the stomach could only be excised by adding total pancreaticoduodenectomy to the radical operative procedure in carcinoma of the stomach.

7. In pyloric carcinoma that remains reasonably localized in the gross, subtotal gastrectomy plus radical excision of the immediate lymphatic drainage is more likely to improve the cure rate than is routine total gastrectomy.

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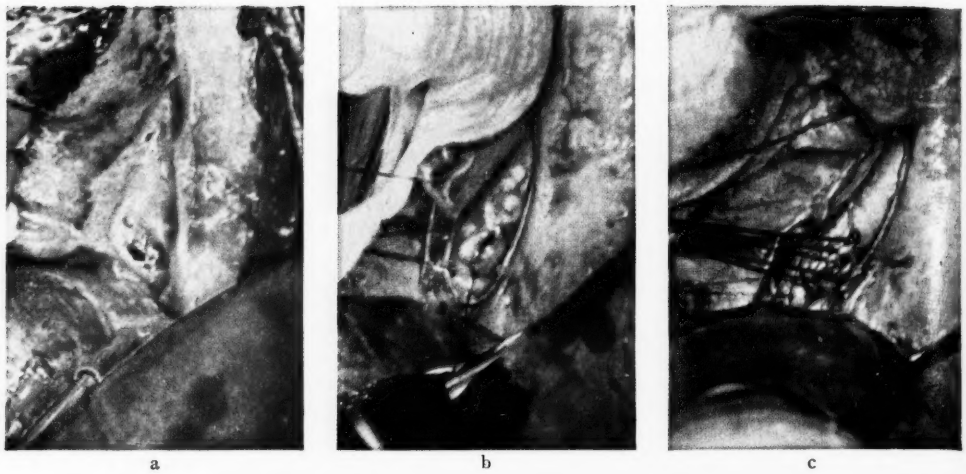


a b c

Fig. 2.

Fig. 3.

Fig. 2. Views taken from a motion picture of the operative procedure in Case 1. a, Note perforation in mediastinal pleura. b, Mediastinal pleura has been incised, exposing esophagus. The rupture in the esophagus extends through diaphragmatic hiatus. c, Rupture has been sutured.

Fig. 3. Specimen showing artificially produced rupture in the esophagus of a fresh human cadaver. The tear was produced by inflating the esophagus with air until it burst. Note the similarity with the lesion which is illustrated in Figure 2.

SPONTANEOUS RUPTURE OF THE ESOPHAGUS

An Experimental and Clinical Study

S. A. MACKLER, M.D., F.A.C.S., Chicago, Illinois

SPONTANEOUS rupture of the esophagus is an uncommon accident which occurs, in most instances, following prolonged or forceful vomiting. The rupture is characterized by a sudden splitting apart of the wall of an apparently normal esophagus with the escape of gastric content into the mediastinal space.

It is necessary to distinguish between rupture of the esophagus and perforation of the esophagus. Perforation, except when produced by direct trauma, usually instrumentation, is the end result of a disease process involving the wall of the organ during which there is constant and progressive erosion. The perforation appears as a small, round hole and shows no predilection of site as to the circumference or axis of the esophagus. In contrast to perforation, rupture occurs as an abrupt tear in an apparently undiseased esophagus. The defect appears as a longitudinal rent and it is almost invariably located at the same site (Figs. 1, 2, 3).

The factors responsible for solution in continuity of the esophageal wall can be outlined as follows:

Perforation:
- Direct trauma (instrumentation, missiles)
- Peptic ulcer of the esophagus
- Esophagitis
- Impaction of a foreign body
- Impaction of debris above a stricture
- Ingestion of caustics
- Carcinomatous erosion
- Erosion by tuberculosis in adjacent lymph node
- Syphilis
- Aneurysm
- Diverticulum

Rupture:
- Indirect trauma
- Spontaneous

A clear distinction is made between perforation and rupture. It is to be noted that rupture is classified as being spontaneous in character or following blunt injury to the lower thorax or abdomen. The relationship between rupture of "spontaneous" and traumatic origin, and the probability of a common modus in the production of the rupture, as intimated by the similar appearance of the lesions, is discussed in the following.

The earliest recorded case of spontaneous rupture of the esophagus was that of Herman Boerhaave of Holland in 1724. From the time of Boerhaave to the present there have been recorded a total of 73 true cases of spontaneous rupture of the esophagus. This excludes those in which the esophagus had undergone perforation rather than rupture, and those in which the character of the lesion could not be determined because of lack of reported data. The disease was uniformly fatal until 1947 when the first case to survive was reported (22). Recovery followed the surgical repair of the esophageal defect. The employment of surgery as a method of treatment had been tried upon only two occasions (6, 10) prior to this time. The apparent reason for faltering in utilizing surgical treatment lay in the failure to establish a premortem diagnosis. It was recognized that recovery from this lethal disease was dependent upon prompt surgical intervention and the only limiting factor was that of early diagnosis (7).

DIAGNOSIS AND MORBID PHYSIOLOGY

A correct diagnosis is possible only if rupture of the esophagus is kept in mind when considering the differential diagnosis of medical emergencies. The clinical picture is that of an adult male who, following a heavy meal, commences to vomit. Shortly following severe or protracted retching, sharp pain is experienced in the lower sternal region of the chest which radiates to the back and localizes in the region of the tenth thoracic vertebra, usually to the left of the midline. The sensation of

From the Thoracic Surgical Service, Cook County Hospital, and the Department of Thoracic Surgery, Chicago Medical School.

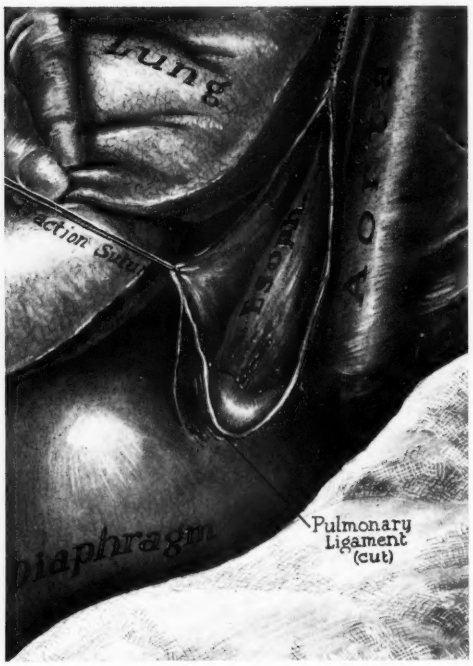


Fig. 1. Artist's drawing of rupture of the esophagus (Case 1). Note longitudinal, linear rent in the left lateral wall of the esophagus. In this instance, the tear extended through the diaphragmatic hiatus to reach the esophagogastric junction.

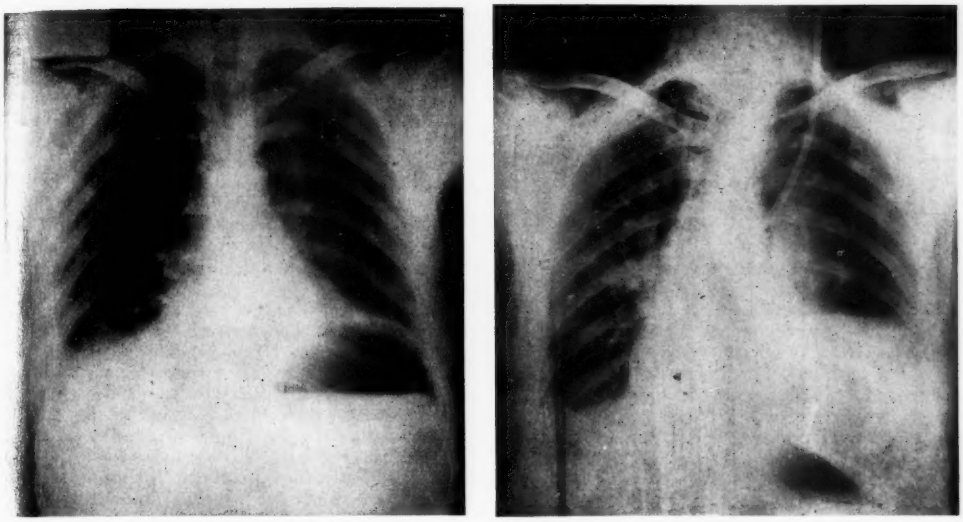
"something giving way internally" may be experienced, and although uncommon, a small amount of bright red blood may appear in the emesis at this time. With the onset of pain, vomiting ceases. There are accompanying signs of collapse: rapid pulse, sweating, cold clammy skin, ashen color, and a lowered blood pressure.

The physical signs early in the course of the disease are disappointingly meager. In many reported cases there are signs referable to the upper abdomen, tenderness and muscular rigidity. Indeed, the usual diagnosis is that of a perforated peptic ulcer, the true nature of the condition first being suspected when no lesion is found at the time of laparotomy. That early physical signs referable to the chest are scant or absent is understandable. Following the tear in the esophageal wall, the liquid, solid, and gaseous contents of the stomach escape to accumulate within the mediastinal compartment encased laterally by the right and the left mediastinal pleurae, anteriorly by the heart, and posteriorly by the vertebral bodies. The detection of an accumulation within the mediastinum by physical examination alone, unless suspected, taxes diagnostic acumen even under the most favorable circumstances. It is only when erosion and perforation of the mediastinal pleura finally take place and the gastric content is spilled into the pleural cavity that attention is focused to the chest. The time necessary for invasion of the pleural cavity is variable and depends upon the resistance offered by the mediastinal pleura, which acts as a barrier. In the case described by Collis, operation was performed 9 hours following the onset of symptoms, yet the mediastinal pleura was still intact. In 1 case described herein (Case 1), roentgenographic examination 4 hours after the onset of symptoms showed the pleural cavities to be uninvolved (Fig. 4a); but re-examination 8 hours later disclosed a left hydropneumothorax (Fig. 4b). Perforation of the mediastinal pleura had taken place during this lapse of time.

Prior to perforation and disgorgement into a pleural cavity, almost invariably the left, the accumulation within the mediastinal compartment, under mounting pressure, dissects along tissue planes. The pathway affording the least resistance is upward, from the crura of the diaphragm to the superior mediastinum and the superior thoracic strait to the tissues of the neck. While the two major bronchi and the hila of the lungs obtrude as obstacles to free passage for a liquid collection in the posterior mediastinum, the gas quickly finds its way around these structures and ascends to the loose areolar tissues of the root of the neck. Crepitus may be detected here within 1 hour after the rupture (Cases 1, 3).

During this interim, too, before pleural perforation occurs, there frequently appears a bilateral pleural effusion of clear fluid. This results from the irritant action of gastric content upon the mediastinal surfaces of both pleurae, stimulating the secretion of fluid within each pleural cavity.

It is evident that the early diagnosis of spontaneous rupture of the esophagus de-



a b

Fig. 4. Case 1. a, Roentgenogram taken 4 hours after set of symptoms. The accumulation of gastric content within the mediastinum is not apparent because of the cardiac shadow. b, Roentgenogram 8 hours later, now showing left hydropneumothorax. The accumulation has perforated the mediastinal pleura (see Fig. 2a) and has spilled into the left pleural cavity. Note emphysema of the neck in each roentgenogram.

pends upon an awareness that there exists such an entity when considering the differential diagnosis of medical emergencies. Suspicion is aroused by the history of acute thoracic pain during vomiting. The disclosure of a collection within the mediastinum crystallizes the suspicion to a certainty. The gaseous (air) component of the mediastinal collection (mediastinal emphysema) gives the first clue and is detected by palpating the crepitus of interstitial emphysema at the base of the neck. Roentgenograms or fluoroscopy may be helpful in confirming the presence of a mediastinal accumulation, but visualization of a fluid level may be obscured by the density of the heart shadow. It is desirable to forestall the development of a pleural complication by early operation. If, however, perforation of the pleura has already taken place, the presence of a hydropneumothorax further substantiates the diagnosis. A small quantity of methylene blue solution, taken by mouth, and appearing in the fluid obtained by thoracentesis offers conclusive proof of the diagnosis. It is to be emphasized, however, that a hydropneumothorax indicates a lapse of time sufficient for pleural perforation, yet early diagnosis and operation is of the essence for a successful result. It seems reasonable to state that, given an acutely ill patient exhibiting signs of collapse, *the elicitation of a history of sudden thoracic pain occurring during or shortly after the act of vomiting, followed by the appearance of interstitial emphysema at the base of the neck, constitutes sufficient evidence to warrant a left thoracotomy.*

CASE REPORTS

The following 3 cases of spontaneous rupture of the esophagus present the history and findings characteristic of this disease. In each instance the diagnosis was made prior to operation. Two of the patients recovered following surgical therapy. The addition of these recoveries to those previously reported in the literature brings to a total of 13 those surviving a spontaneous rupture of the esophagus.

CASE 1. Mr. D. B., aged 47 years, was admitted to the Cook County Hospital on October 29, 1950, at 10:00 p.m. For 3 previous days there had been intermittent midepigastric discomfort and emesis 2 to 3 times per day, occurring soon after eating. At

7:00 p.m., 3 hours prior to admission, there occurred a severe episode of vomiting which was followed by sudden thoracic pain distinctly separate from his previous abdominal discomfort. The pain, which progressively became more intense, was located at the lower sternal area and radiated to the back to the left of the midline. Retching movements continued but they were unaccompanied by emesis. The patient volunteered the information that his "neck became swollen" within 1 hour after the onset of pain. On examination the patient appeared to be acutely ill, but not in critical condition. The temperature was 100.6 degrees F., respirations 24 per minute, pulse rate 104, and the blood pressure 105/70. The white blood cell count was 18,200. The only abnormal finding was emphysema of the soft tissues of the neck. Roentgenologic examination of the chest and abdomen showed no abnormalities, but air was seen in the fascial planes and the subcutaneum of the neck (Fig. 4a).

A diagnosis of spontaneous rupture of the esophagus was made at this time. Because of the occurrence of an unusually large number of cases requiring emergency surgery, operative treatment was delayed until 10:00 a.m. of the following morning, approximately 15 hours following the rupture. A roentgenogram taken at 7:00 a.m. October 30 now showed the presence of a left hydropneumothorax (Fig. 4b). The gastric contents had perforated into the left pleural cavity. The patient's condition had deteriorated during the delay; the pulse rate was 120 and thready in quality, the blood pressure 90/60.

A thoracotomy was performed and the left pleural cavity entered via the bed of the resected eighth rib. The pleural cavity contained approximately 2,000 cubic centimeters of murky brown fluid in which floated small particles of food debris. This issued from an oval hole in the mediastinal pleura at the base of the pulmonary ligament (Fig. 2a). The entire mediastinal pleura was incised, exposing the esophagus. The left lateral wall contained a longitudinal rent which disappeared with the esophagus through the hiatus of the diaphragm. By retraction of the margins of the hiatus and by upward traction of the esophagus, it was possible to expose the inferior extremity of the tear which extended for a distance of 2 inches to reach the cardia of the stomach (Figs. 1, 2b). A biopsy of the mucous membrane was taken from the opposing lips of the defect (Fig. 7a). There was no ulceration or necrosis of tissue visible at this site. The rent was closed in two layers, mucous membrane and muscularis, using interrupted sutures of No. 0000 silk (Fig. 2c). Crepitus could be felt beneath the pleura overlying the aorta. This was incised and the aorta laid bare, thus freeing all loculations to drain into the pleural cavity. The chest was then closed after the insertion of 2 intercostal tubes to drain the pleural cavity by water-seal.

The postoperative recovery was surprisingly uneventful and he was discharged from the active surgical ward 2 weeks following operation. Roentgen visualization of the upper alimentary tract 3 weeks following operation showed a normal esophagus, but there was marked deformity and spasticity of the duodenal bulb. This, evidently, was the element originally responsible for the emetic episodes.

CASE 2. Mr. R. R., aged 32 years, was admitted to the Cook County Hospital at 1:00 a.m. March 20, 1950. He had not had any previous alimentary disturbance until the preceding evening, when, after a late dinner at which he had overindulged his appetite, he became ill. Attempts were made to induce vomiting by inserting a finger in his throat. He was finally rewarded by a series of violent vomiting episodes. In the midst of a spasm of severe retching, he was suddenly seized with a sharp, knife-like pain in the lower sternal area which radiated to the left side of his back, and he collapsed.

Upon admission to the hospital 1 hour later he appeared acutely ill. His skin was cold and ashen in color, pulse rate 120, and blood pressure 80/40. There was tenderness and spasticity in the region of the upper abdomen. The impression was that of a perforated peptic ulcer. A Levin tube was inserted and plasma and dextrose solutions were given intravenously. His condition improved and while his abdomen at no time exhibited a board-like rigidity, the spasticity now disappeared leaving a soft abdominal wall. The blood pressure rose to 100/80 and the pulse rate remained at 100.

A roentgen film of the abdomen showed no subdiaphragmatic air. Operation upon the abdomen was deferred. His condition remained stationary throughout the next morning and early afternoon. A roentgenogram of the chest taken at 5:00 p.m. showed clouding of the left lower hemithorax. Fluoroscopy of the chest was done later and it appeared that a fluid-level was present behind the cardiac shadow. Crepitus was now sought at the base of the neck, and detected. The diagnosis of spontaneous rupture of the esophagus was now made.

The patient's condition began to deteriorate rapidly, and at the time of operation at midnight, March 20–21, twenty-four hours following the rupture, his status was desperate. A left thoracotomy was done through the bed of the resected eighth rib. The left lung was adherent to the parietal pleura by numerous adhesions. Upon severing these a turbid brown fluid was encountered in loculations which originated from a reservoir at the base of the pulmonary ligament. This, in turn, was fed through a small hole in the mediastinal pleura, which was tense and bulging. At this time the blood pressure was 30/0 and the pulse could not be obtained. Upon incising the mediastinal pleura there escaped under pressure a large quantity of the fluid containing quantities of food debris. Hamburger meat, onion, and cheese were recovered from the mediastinum and the surface of the pericardium and other visceral structures. There was noticed an immediate response in the patient's condition, which rapidly improved.

The fluid and the food content issued from a longitudinal rent in the lower end of the thoracic esoph-

agus. The split appeared in the left lateral wall, extended for a distance of 1½ inches and ended approximately ½ inch above the diaphragm. The margins of the wound were clean and there was no evidence of necrosis or slough which might have preceded the rupture. The mucous membrane and the muscle were closed separately with interrupted sutures of No. 0000 silk. The entire length of the mediastinal pleura as well as that overlying the aorta was incised widely to allow free drainage of secretions into the pleural cavity. Two intercostal tubes were then inserted into the pleural cavity for the purpose of closed drainage prior to closing the chest.

The immediate postoperative course was stormy, with temperature elevations to 105 degrees F. With the help of antibiotics, blood transfusions, and the intravenous administration of solutions for the maintenance of metabolic and electrolyte requirements, the patient began to improve on the fifth postoperative day. His recovery progressed rapidly thereafter and he was discharged from the hospital 3 weeks from the time of operation. A later roentgenologic examination of the esophagus and upper intestinal tract showed no abnormalities.

CASE 3. Mr. J. B., aged 51 years, entered the Cook County Hospital at 10:00 a.m. November 11, 1951. He had had no alimentary disturbances until the day prior to admission. He complained of "indigestion" during the day, and at 6:00 p.m., during dinner, he became nauseated and started to vomit. During the third episode of vomiting, he experienced a sharp substernal pain, and became weak and faint. Bright red blood was seen in the emesis. It was noted that his neck became "swollen" 1 hour later. The pain persisted throughout the night, and he applied for admission to the hospital at 10:00 a.m. the following morning.

The patient's condition upon entrance was critical; pulse rate 130, blood pressure 90/60. Signs of peripheral vascular collapse were evident. He was given plasma and dextrose solution intravenously. The only positive physical finding was that of subcutaneous emphysema of the neck. A roentgen film showed the presence of a small effusion in the *right* hemithorax; the left hemithorax was normal in appearance (Fig. 5). A diagnosis of spontaneous rupture of the esophagus was made.

Operation was performed at 3:00 p.m. A left thoracotomy was performed in spite of the fact that it was the right pleural cavity which contained fluid. The left pleural cavity was entered after rapidly resecting the eighth rib. At this point the blood pressure was 30/0 and the patient's condition was critical. In addition to the intravenous administration of citrated blood, an intra-arterial blood transfusion was now started by way of the left radial artery. The left lung was adherent to the parietal pleura by numerous adhesions. A small amount of brownish colored, free fluid was present. The mediastinal pleura was tense and bulging. Upon incising this there escaped under pressure a large quantity of

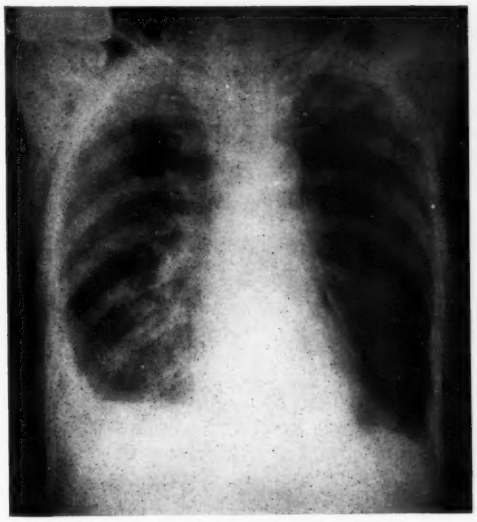


Fig. 5. Roentgenogram in Case 3 showing fluid in the right pleural cavity. A left thoracotomy was performed, even though the left hemithorax is clear, and the rupture found in the left wall of the esophagus. Note emphysema of the neck extending to the subcutaneum of the thoracic wall.

brown turbid fluid containing food debris. This issued from a longitudinal rent in the left lateral wall of the esophagus. The tear measured 2 inches in length and extended through the diaphragmatic hiatus to reach the cardia of the stomach. A biopsy was taken from the margin of the rent (Fig. 7b). The defect was sutured in layers, mucous membrane and muscularis, using sutures of No. 000 silk. The entire length of the mediastinum was then laid open for free drainage into the pleural cavity. The tissues appeared to be in an advanced state of digestion. The chest was closed after inserting 2 tubes for closed drainage of the pleural cavity.

The patient rallied soon after decompression of the mediastinum, and he left the operating room with a blood pressure of 100/70. His circulation began to falter again later, however, and in spite of bolstering it with plasma and blood transfusions, he expired at 9:00 p.m. Permission for a postmortem examination was not granted.

ETIOLOGY

The emergency is predominately a male disorder. Ridgeway and Duncan collected 35 cases from the literature and added 1 of their own. There were only 5 females in this series. The preponderance of males in approximately the same ratio is substantiated by subsequent

case reports. This may represent a sex predisposition but, more likely, as Barrett suggests, a male inclination to indulge in pursuits which predispose to vomiting.

While rupture of the esophagus has been reported in all age groups, the youngest in an infant of 5 months (19), most patients have been in the fourth or fifth decades of life.

There is no unanimity of opinion as to the cause of the rupture although many explanations have been proposed.

Boerhaave attributed rupture due to direct traction on the esophagus, the lower end of the tube being drawn downward by the weight of the overloaded stomach, aided by the contraction of the diaphragm, while the superior extremity was forcibly stretched above by straining efforts and the forward movement of the hyoid.

Murdoch, in commenting upon a rupture which followed trauma to the abdomen, suggests a sudden stretch of the diaphragm at the hiatus with a tugging effect upon the esophagus and resultant rupture.

Mallory and Weiss suggest alcoholism as a necessary predisposing factor. They have demonstrated linear ulcerations in the mucous membrane at the cardia and suggest that this precedes rupture. They point out that pressure changes during the disturbed mechanism of vomiting cause fissures in the mucous membrane at the cardia, and the local irritant, astringent, and corrosive effect of alcohol, plus the regurgitated gastric content, result in linear ulcerations. (Alcoholism was present in 18 of the 35 cases of Ridgeway and Duncan.)

Hoffman (12, 13) and Zenker and Ziemssen consider esophageal malacia, a softening of the coats of the tube. It is conceived that repeated efforts at vomiting cause regurgitated food and gastric juice to remain in the gullet sufficiently long to give rise to digestive softening of its walls.

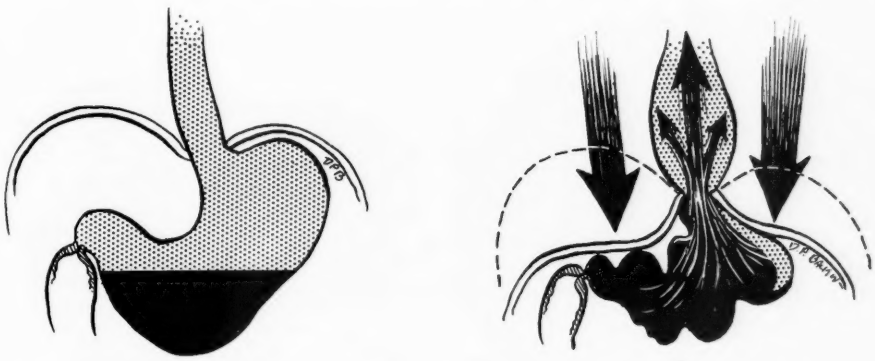
Williams and Boyd suspect that pre-existing inflammation with inflammatory softening and round cell infiltration at the site to be a predisposing cause.

Collis, Humphreys, and Bond conjecture that intragastric pressures produced during vomiting may be sufficient to cause rupture of the esophagus.

In an attempt to account for the etiology of rupture of the esophagus, the records of previously reported cases were scrutinized for a common factor. The act of vomiting stands out as the prevalent theme. Isolated cases have occurred, however, in which vomiting has not been associated with a spontaneous rupture of the esophagus. A clue to etiology is provided by these instances, for, while vomiting has been absent, there occurred other acts in which there was produced a sudden rise in intra-abdominal pressure. Thus, rupture has occurred while straining at defecation (31), during the effort of lifting a 50 pound weight (11), as a result of a convulsive seizure of grand mal epilepsy (16), and during the labor of childbirth (14).

A sudden rise in intra-abdominal pressure also occurs during the act of vomiting. Vomiting is a series of co-ordinated reflexes, the expulsive force, however, being supplied by convulsive abdominal compression and not, as might be supposed, by antiperistaltic activity of the esophageal tube (3). Indeed, the role of the esophagus and stomach is purely passive in nature. A deep contraction occurs in the region of the incisura, clamping shut the pyloric end of the stomach. The upper portion of the stomach, or cardiac pouch, and the cardiac sphincter undergo complete relaxation. Prior to the commencement of vomiting the reduction in gastric tone is such that the stomach drops several inches in the abdominal cavity, as seen roentgenographically. The esophagus, too, is relaxed throughout. The hyoid bone and the larynx are drawn and held forward, thus enlarging the opening of the pharyngo-esophageal region to allow free exit for the stomach content. A sudden increase in the intra-abdominal pressure is then effected by a sharp descent of the diaphragm and contraction of the abdominal muscles. The dilated, patulous stomach is now subjected to a strong compressive force and the gastric content is forcefully ejected through a relaxed cardia into the esophagus (Figs. 6a, b).

It seems logical to conjecture that the direct application of force to the abdomen by external violence, causing a sudden rise in intra-abdominal pressure, might also result in a rupture of the esophagus. Actually such in-



a b

Fig. 6. Diagrammatic representation of the mechanism vomiting. a, Inhibition of tone and flaccid relation of e cardiac pouch and sphincter of the stomach. b, Sharp contraction of the diaphragm and abdominal muscles with resultant compression of the stomach and forcible ejection of its contents into the esophagus.

idents have been reported. Rupture of the sophagus has occurred as a result of having een struck across the upper abdomen and wer thorax by a falling mantlepiece (1), run ross the abdomen by a motorcycle (21), and inned underneath an automobile (23). To hese may be added a personal case, not previously reported.

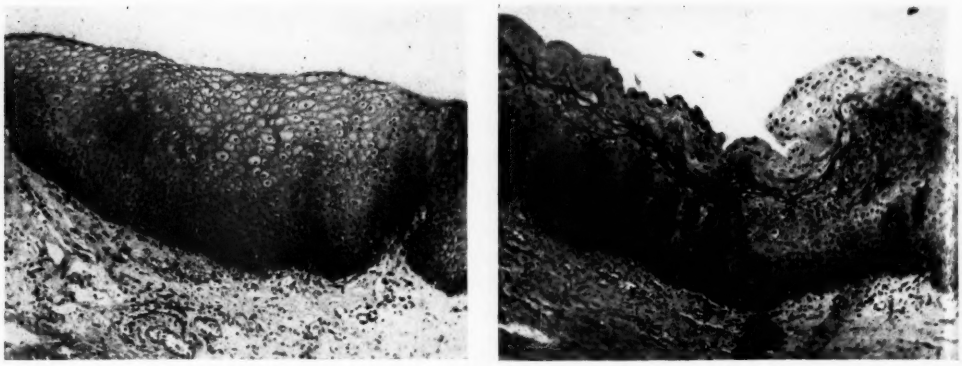
The incident happened in 1944 during the period of the Italian campaign of World War II.

A British soldier, a motorcycle courier, was thrown from his vehicle and fell in a prone position across a milestone at the roadside. The impact was taken by the upper abdomen. A roentgen film of the chest taken 2 hours following the injury showed several rib fractures, but both lungs were fully expanded and there was no fluid or air in either pleural cavity. His condition became unaccountably worse and he died 12 hours later, 14 hours following the injury. A postmortem examination now revealed air and gastric contents in the left pleural cavity which had issued from a rent, 1 inch in length, in the left lateral wall of the esophagus. Its location was immediately above the diaphragm. There were no bony fragments in this region which could possibly have lacerated the esophagus.

With 1 exception, the site and configuration of the esophageal tear were the same in all cases in which the rupture followed blunt trauma to the abdomen, and these, in turn, duplicated the pattern where rupture occurred spontaneously, following vomiting or other acts involving a sudden rise in intra-abdominal pressure.

It appears, then, that the forces exerted upon the stomach and esophagus are essentially the same whether generated by the act of vomiting, by sudden mechanical compression, as in blunt trauma to the abdomen, or by any other act by which a sudden rise in intra-abdominal pressure is produced (straining at defecation, lifting a heavy weight, convulsive seizure, labor of childbirth). A full stomach would seem to be a necessary condition, however, if rupture of the esophagus is to take place by the transmitted force of the gastric content. The history that rupture occurs shortly after ingesting a large meal is a prominent feature in the records of published case reports. Thus, in Griffith's case, rupture followed lifting a heavy weight immediately after eating a large meal; in Klein and Grossman's report, the accident occurred during a convulsive seizure which took place following a heavy meal. In the group of cases in which rupture resulted from blunt injury, there is no mention of a previous large repast, but it is justifiable to make such an assumption because of the quantity of gastric content found in the pleural cavity. When vomiting has been associated with rupture, the ingestion of a large quantity of food prior to the accident has been reported almost routinely.

It seems reasonable to contend then, that vomiting is the cause and not the effect of spontaneous rupture of the esophagus; that the sudden rise in intra-abdominal pressure



a b

Fig. 7. Photomicrograph of biopsy of the mucous membrane taken from the margin of the rupture which occurred in Case 1 (a) and in Case 3 (b). There is no visible necrosis of tissue and no evidence of pre-existing ulceration. The presence of capillary dilatation and of small hemorrhagic areas in the submucosal layer indicates only acute reaction which is compatible with the trauma of rupture (×50).

produced by retching, as well as when produced by other means, violently ejects gastric content into the esophagus by compression of the stomach; that the sudden rise in intraluminal pressure of the esophagus may be sufficient to overcome the tensile strength of the wall of the tube, which then bursts.

Is it possible to generate a force sufficient to burst a normal, healthy esophagus by the effort of vomiting? An affirmative answer is suggested by the fact that rupture of the esophagus has occurred in numerous cases in which vomiting was stimulated by causes unassociated with this organ, and in which there was no history of any pre-existing esophageal disease. Thus, rupture has occurred during the vomiting caused by seasickness (28), anesthesia (26), small intestinal obstruction (29), cerebral stimulation (8), self-induction (4, 5), peritoneal irritation (carcinomatosis and ascites) (18), and large ovarian cysts (18).

The appearance of the esophagus at the margin of the tear usually shows no pathologic change concomitant with a pre-existing ulceration. In some instances moderate lymphocytic infiltration of the contiguous mucosa and submucosa has been described. Whether this indication of chronicity can be construed as evidence of a pre-existing inflammatory state is difficult to state. In the normal esophagus removed at necropsy the number of scattered lymphocytes and lymphoid follicles vary considerably. In most instances, the inflammatory changes are acute and readily explained upon the basis of the trauma itself. Biopsies of the mucous membrane taken from the site of the tear during the surgical procedures in Case 1 (Fig. 7a) and Case 3 (Fig. 7b) confirm this; namely, that the inflammation is acute and attributable to the trauma of rupture itself, rather than existent prior to the time of the rupture so as to have altered or weakened the wall of the esophagus where the tear had taken place. The shape of the rent, too, suggests force rather than ulceration and digestion. If the latter, one would anticipate a more or less circular punched out perforation, or at least an irregular patch of partially digested sloughing tissue in the wall of the esophagus rather than the clean cut longitudinal slit that is found (25).

The query may be raised as to whether sufficient pressure could be built up within the esophagus if there exists no obstruction toward the outlet of the tube. It is difficult to conceive that pressure could be mounted to a degree sufficient to bursting in a freely patent gullet. Organic obstruction to the free egress of vomitus has been described in many cases in which rupture has occurred. In the case reported by Meyer, a fragment of sausage became impacted in an esophagus affected by an old lye stricture. Repeated vomiting in attempting to dislodge the bolus resulted in

rupture of the *normal esophagus below* the site of impaction. In Fitz's case, rupture followed attempts to dislodge an impacted bolus of meat by repeated vomiting. There is the case of a child, aged 5 months (19), who had been prone to attacks of emesis, ascribed to overfeeding. During one of these episodes "a curd of milk became stuck in the throat" and further vomiting to dislodge the obstruction resulted in rupture. At postmortem examination there was no evidence of any previous organic disorder nor of pre-existing inflammation.

Rupture has occurred in the lower esophagus when only an incomplete stricture was present at the oral end. Although complete obstruction was not effected by impaction of food matter, as in the previous episodes, it seems evident that an adequate avenue of escape could not be yielded quickly enough to decompress the sudden inflation of the esophagus from below.

The majority of cases of rupture of the esophagus, however, show no demonstrable obstruction. Yet, if the concept is to obtain that the hydrodynamic forces concomitant with vomiting are responsible for rupture of the esophagus, an explanation must be provided for the occurrence of rupture where no obstruction can be demonstrated. It is entirely possible that an obstructive element may be present, but in a functional rather than an organic manner, in the nature of sphincteric spasm. The act of vomiting is executed by a series of co-ordinated reflexes amongst which there occurs a forward movement of the larynx and hyoid bone and a relaxation of the cricopharyngeal sphincter to provide a spacious exit for the gastric content. In prolonged and repeated vomiting, ensuing fatigue of the vomiting center or fatigue of the muscular components with failure to respond promptly to nervous stimuli may result in inco-ordination. Reflex relaxation of the upper outlet may fail to coincide with the onrush of gas and liquid content from the stomach. The presence of previously regurgitated hydrochloric acid in this region too, may promote spasm and delay in prompt relaxation. The sphincteric obstruction in the face of the generating forces from below may be responsible for the momentary development of sufficient pent-up pressure to burst the esophagus.

EXPERIMENTAL DATA

If this mechanism is the responsible one for creating the tear, there should be an equal distribution of internal stress throughout upon the walls of the esophagus, yet the rupture almost invariably occurs at a selected site. One must presuppose, therefore, that the region in question is anatomically the weakest part of the esophageal wall, and hence the first to give way under strain.

To determine the correctness of this assumption, the esophagi of 65 fresh human cadavers were inflated to bursting to determine (a) whether a tear so produced would occur with uniformity as to site and configuration, (b) whether the tear would conform to the rupture as found clinically, and (c) the tensile strength of the esophageal wall.

Cadavers were selected which were free of disease of the upper alimentary tract. The esophagus and the stomach were removed in continuity as soon after death as possible to prevent autolytic digestion and digestion by the gastric juice. The lower end of the esophageal tube was occluded by encircling the cardia of the stomach with a stout ligature immediately below the esophagogastric junction. The outlet of an ordinary automobile tire pump, to which was attached a manometer, was tied into the oral end of the esophagus, and the organ then infiltrated with air until it burst.

There were 24 female and 41 male cadavers from whom the esophagi were removed. The ages at time of death ranged from 24 to 80 years, with an average of 56.6 years. The lapse of time from death to removal and inflation of the esophagi varied from 1 hour to 31 hours, with an average of 12.0 hours.

The pressure required to burst such esophagi ranged from 4.5 pounds to 7.4 pounds, with an average of 5.0 pounds.

With 3 exceptions, all the tears so produced occurred at the lower extremity of the esophagus, in the left lateral wall (Fig. 3) and duplicated exactly the lesion in which the rupture occurs clinically (Figs. 1 and 2). All were longitudinal linear rents and extended from

½ inch to 1¾ inches (average 0.95 in.) in length. The inferior limit of the tear reached from ½ inch to 1½ inches above the esophagogastric junction (average 0.87 in.).

Of the 3 exceptions, 1 occurred in the upper half upon the anterior wall. The remaining 2 appeared at the lower extremity of the esophagus, but, while reflecting the image of the usual rent as described, occurred upon the right lateral wall. All tears were longitudinal.

It is of interest to note that the mucous membrane is the last coat to burst upon inflating the organ. The muscularis first splits in the direction of the axis of the tube, and a bubble of mucous membrane balloons out through the aperture. This then bursts.

These data confirm the findings of Mackenzie, who, in 1880, carried out a similar series of experiments. Esophagi were removed from 18 fresh cadavers, and water forced in under pressure. In 16 cases, rupture occurred from 2 to 3½ inches above the cardia and resembled entirely the lesion found in spontaneous rupture. In the 2 cases in which the tear did not conform strictly to this measured distance, the rupture occurred, nevertheless, in the lower half. In no case did a split appear in the upper half of the esophagus. The extent of the tear varied from ⅓ inch to 2 inches in length. In all instances, the solution in continuity was in a longitudinal direction. The average pressure required to burst the esophagus was 7 pounds.

There are two objections which may be raised against these experiments; namely, that the specimens may have undergone some degree of postmortem digestion, particularly at the lower end of the esophagus because of its proximity to the gastric juices; and that by removing the esophagus from its bed one removes the buttressing effect of contiguous supporting structures (aorta, etc.). To these objections may be cited an accidental in vivo experiment (24). A man occluded a compressed air tube with his hand and placed the end in his mouth as a gesture of mirth. He accidentally slipped and let go of the tube. The resultant gush of air down the esophagus burst the organ in the lower one-third. Postmortem examination also disclosed involvement of the left pleural cavity.

In seeking to account for the weakness of the lower esophagus as compared to the upper portion, several explanations seem plausible. Histologically, the quality of the musculature differs at various levels. The upper one-third contains exclusively cross-striated muscle fibers, like those of the pharynx. Below this there is a zone of intermingled smooth and cross-striated fibers. In the lower one-third, however, the muscle is composed exclusively of smooth fibers. Secondly, the upper one-half of the esophagus, in situ, is further strengthened by the trachea, which is in intimate contact with its anterior wall. A third factor, which may be of some account, is the splaying out of the muscle fibers of the lower extremity of the esophagus as they begin to spread to form a broad coat for the stomach.

TREATMENT

Prompt surgical intervention had long been recognized as the treatment offering the only opportunity for a successful outcome of this heretofore consistently lethal disease. The first successful result (22) followed exposure of the esophagus and suture of the defect, by way of a left thoracotomy, and drainage of the pleural cavity.

Surgical repair of the esophageal lesion has been performed in a total of 14 cases. The approach was by way of a left thoracotomy in each instance except one, in which the esophagus was drawn into the peritoneal cavity through the hiatus of the diaphragm during an abdominal exploration. There were 9 patients who recovered. In each case in which the esophagus was successfully repaired, the approach was by way of a left thoracotomy.

Drainage of the pleural cavity or mediastinum alone, without attempting to repair the esophagus, has also been done as a method of surgical treatment. It is extremely difficult to determine the true nature of the leak as the esophagus is not exposed or visualized in such cases. In most instances the reason for instituting drainage was the presence of an abscess of the mediastinum or pleural cavity (empyema). The development of such a suppurative process is more likely to follow a perforation rather than a rupture of the esophagus. The more chronic process of erosion, preparatory

to final perforation, is attended by an inflammatory process which involves the contiguous structures. Hence, at the time of perforation the regional tissues, as a result of preceding inflammation, are adherent and prepared to localize the spillage. The end result, then, is the abscess. In contrast, rupture occurs abruptly in a previously undiseased esophagus. There exists no preceding inflammatory process to prepare the periesophageal tissue for containment. The sudden spillage of gastric content into the virgin tissue spaces of the mediastinum occurs before any attempt at localization is accomplished, and the result is free inundation, rapid absorption of toxic products, and collapse of the patient. The tempo is too rapid for the development of an abscess.

Careful examination of the records indicates that of those patients treated by drainage alone, 6 may be accepted as authentic cases of spontaneous rupture of the esophagus. Of these, 2 recovered, a mortality of 66.6 per cent. In comparison, 16 patients (including the 3 herein reported) were treated by thoracotomy, definitive repair of the esophageal lesion, and drainage of the pleural cavity. Of these, 11 recovered, a mortality of 31.3 per cent.

It seems clear that the treatment of spontaneous rupture of the esophagus is prompt surgical intervention. The preferred operative procedure consists of a left thoracotomy, exposure of the esophagus, and suture of the defect. The mediastinal pleura is incised throughout its entire length to decompress all loculated areas within the mediastinum and allow free drainage into the left pleural cavity. The pleural cavity is then drained by means of a closed drainage system.

SUMMARY AND CONCLUSIONS

1. Spontaneous rupture of the esophagus is an accident which may follow a sudden rise in intra-abdominal pressure, usually vomiting. Other acts which have resulted in rupture are: straining at defecation, lifting a heavy weight, a convulsive seizure of grand mal epilepsy, and the labor of childbirth.

2. Distinction is made between rupture and perforation of the esophagus.

3. The triad of vomiting, low thoracic pain, and emphysema of the neck, is diagnostic and constitutes sufficient evidence to warrant a left thoracotomy.

4. Evidence is presented to support the conclusion that rupture occurs in a previously normal, healthy esophagus as a result of intra-esophageal pressure, transmitted from the stomach. The rupture uniformly occurs at the lower extremity of the esophagus upon the left lateral wall, apparently the weakest part of the organ. That this is indeed the weakest part of the esophagus was confirmed by experimentally inflating and bursting the esophagi of 65 fresh human cadavers. The site and configuration of the resulting rupture duplicated exactly the clinical lesion.

5. Three cases of spontaneous rupture of the esophagus are presented. The treatment was surgical: left thoracotomy, suture of the esophagus, and drainage. The recovery of 2 of the patients brings to a total of 13 those surviving a spontaneous rupture of the esophagus. The outcome was uniformly fatal before the adoption of a surgical method of treatment.

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A PHYLOGENETIC STUDY OF RECURRENT ANTERIOR DISLOCATION OF THE SHOULDER JOINT

JAMES A. DICKSON, M.D., F.A.C.S., Cleveland, Ohio, and
HARRY W. O'DELL, M.D., Akron, Ohio

RECURRENT dislocations of the shoulder have been the object of considerable clinical interest since ancient times. Hippocrates described the symptoms of dislocation of the shoulder and recommended a method of preventing recurrences by producing scarring with a cautery. No additional progress of significance was made in the treatment of this condition until late in the nineteenth century, when the pathologic structural changes observed in such dislocations became known.

Roentgenologic and postoperative studies of the structural changes have led to numerous conflicting theories regarding the pathogenesis of recurrent anterior dislocation of the shoulder and to various operative procedures designed to correct it. The pathologic changes most frequently described are (1) anterior detachment of the glenoid labrum from the bone margin of the glenoid, associated with some degree of stripping of the anterior portion of the capsule from the front of the scapular neck and (2) a defect or flattening of the posterolateral aspect of the articular surface of the head of the humerus. Bankart (2, 3) and his protagonists have insisted that the anterior detachment of the glenoid labrum is the "essential" lesion; and there are others, for example, Palmer and some of his Scandinavian colleagues, who believe that the compression fracture of the head of the humerus should be regarded as the "essential" lesion. Still others, such as Adams, say that either type of lesion alone may predispose to recurrent dislocation, and that when both types of lesion are present in the same shoulder, the tendency to redislocation is very great.

Within the last few years, it has become increasingly evident that the cause of recurrent dislocation of the shoulder cannot be satisfactorily explained nor understood completely on the basis of these pathologic structural changes alone, and there is an increasing awareness that these are more probably the result than the cause of the recurrent dislocations. This opinion has had substantial confirmation in the researches of DePalma, on a large series of "normal" shoulder joints. He pointed out that capsular changes, labral lesions, and humeral head defects were noted in association with aging, but that they also may be produced or aggravated by trauma. DePalma believes that neuromuscular imbalance, chiefly of the short rotator muscles, is the most important single causative factor in redislocation of the shoulder. Codman, Magnuson (12), and Henderson also have stressed the importance of muscular imbalance in these recurrent dislocations.

Facts which favor this viewpoint are: (1) the syndrome is most common in young muscular individuals; (2) there is a definite familial tendency toward recurrent dislocation of the shoulder, and frequently it is bilateral; (3) it is often present in patients with epilepsy and other convulsive states; (4) surgical procedures which limit or control external rotation (no matter what the technique used) have succeeded in preventing redislocation. The foregoing facts are agreed upon by all who have studied the problems presented by recurrent dislocations in the shoulder joint.

The present study was initiated in an effort to go beyond and behind and beneath this area of agreed facts, with the hope of gaining more knowledge as to why and how the tendency to recurrent anterior dislocations develops and to analyze more carefully the mechanical factors involved in the functional imbalance. Since recurrent dislocations are often present in several members of the same

From the Cleveland Clinic, Cleveland, Ohio, and the Akron Clinic, Akron, Ohio.

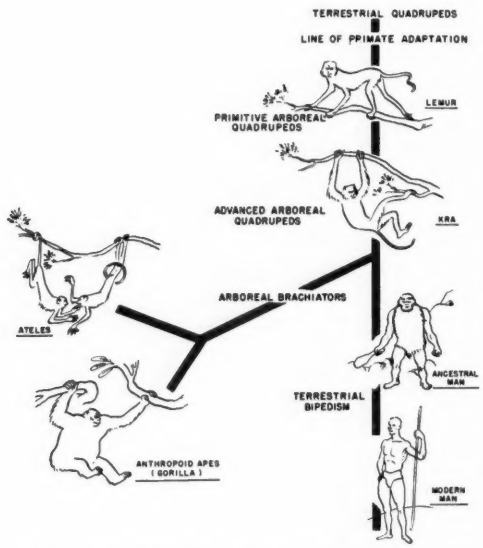


Fig. 1. A modern concept of phylogenetic development of man.

family and since they also are sometimes bilateral, it seemed justifiable to search for some possible developmental anomaly that might account for the clinical data. This led to a study of the shoulder from the phylogenetic, ontogenetic, and dynamic aspects.

PHYLOGENETIC ASPECTS

At one period in the evolution of man, his ancestors walked on all fours on the ground. In accordance with some modern concepts of evolution, certain descendants of these terrestrial quadrupeds jumped into the trees, still using all four extremities for locomotion. The lemur is an example of this primitive type of arboreal quadruped (Fig. 1). A more advanced arboreal quadruped is the old world monkey (kra); this animal always maintains his head erect and never hangs by his tail.

At or near this stage in the evolutionary process, a definite branching occurred. The new world monkey (Ateles) and the great apes, including the chimpanzee, the gorilla, the orangutan and the gibbon, developed from one branch; these animals became brachiators, using their arms for ambulation. The new world monkey also has a prehensile tail and may hang from it or by either his fore or his hind feet. The predecessors of modern man branched off in another functional direction and became terrestrial bipeds. The spine and lower extremities developed for ambulation, while the shoulders and arms were free to develop a greater range of motion for other functions. Since none of the ancestors of man passed through a stage of brachiation, there is no reason to compare him with the new world monkey, the chimpanzee, the gorilla, the orangutan or the gibbon. However, the lemur and the old world monkey are more remote types through which man, in his development, has passed.

When the osteology of the shoulder is compared in the lemur, the old world monkey, and man (Fig. 2), it is evident that, in the process of development, the scapula has increased in length longitudinally, thus giving greater purchase for the short rotator muscles and thereby increasing the rotatory power. The thorax has broadened transversely and has become flatter from the anteroposterior aspect. Concurrently the scapula has moved posteriorly. These changes have increased the range of rotation of the shoulder, allowing man to move his arms in front and behind his body. In the earlier forms, the glenoid faced directly forward. As the evolutionary process advanced, the glenoid has moved in a lateral direction to face both anteriorly and laterally. To accommodate this change, the humeral head has undergone torsion, as a result of which the bicipital groove has moved medially and encroaches on the lesser tuberosity. The combination of the outwardly directed glenoid and the inwardly directed humeral head produces a shoulder capable of universal mobility, but this is achieved at the expense of stability.

Comparison of the shoulder musculature of the lemur, old world monkey, and man (Table I, Figs. 3, 4, and 5) shows that, with more advanced development, the short rotators, excepting the supraspinatus, have increased in size. The supraspinatus has not enlarged, probably because man no longer has the need for overhead power. In the lemur, the pectoralis major and the pectoralis minor are combined in a "common pectoral" mass with

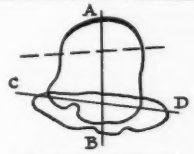
	Scapula	Thorax	Influence of evolutionary torsion upon the tuberosities	Evolutionary turning of humeral head Left humerus
emur	Increased length over earlier mammals	Widens in comparison with earlier mammals	Tubercles of equal size and bicipital groove lies between	 *A-B* Axis of head and neck *C-D* Transv. axis, Epicond.
ld world monkey	Increases in cephalocaudad direction	Continues to widen	Bicipital groove moves medially	
an	Continues to lengthen	Greatest width	The lesser tubercle becomes smaller as the groove moves medially	

Fig. 2. Osteology of the shoulder compared in the lemur, the old world monkey, and man (diagram of the turning of the humeral head from the original of Plate 10, by Ruth A. Miller, *Am. J. Phys. Anthrop.*, 1932, 17: 56).

insertion onto the humerus. A pectoralis abdominis is also present. In the old world monkey, the pectoralis major and the pectoralis minor are separate muscles; however, the pectoralis minor inserts onto the humerus. The pectoralis abdominis is still present. In man, the pectoralis major and the pectoralis minor are distinct muscles; the insertion of the pectoralis minor has moved to the coracoid. The pectoralis abdominis has disappeared.

ONTOGENETIC ASPECTS

An example of the fact that "ontogeny recapitulates phylogeny" is seen in Figures 6 and 7, which show the development of the shoulder in a 9 millimeter and an 11 millimeter embryo, respectively. In the former human embryo, the pectoralis minor and the pectoralis major are combined in a "common pectoral mass" and insert onto the arm. In the larger embryo, the pectoralis minor has begun to delineate as a separate muscle and is moving toward the coracoid.

While these changes are occurring in the pectoral muscle mass, the entire arm bud is undergoing external rotation. At the same time, torsion is progressing at the upper end of the humerus. It is important to recall here the correlation between the external rotation of the limb bud and the internal torsion of the humerus. The rotation of the limb bud in the embryo consists of turning 90 degrees in a lateral direction. The torsion represents the difference in the relative position of the long axis of the proximal and distal ends of the bone and takes place almost entirely at the upper end of the humerus. The primary or inherited internal torsion amounts to approximately 42 degrees, which is that seen in an embryo of 20 weeks. There is relatively rapid change in the degree of torsion throughout the remainder of embryonic life, so that at birth it has reached approximately 65 degrees (Fig. 8). Torsion continues until puberty, but at a greatly decreased rate, and reaches a maximum of approximately 74 degrees at the

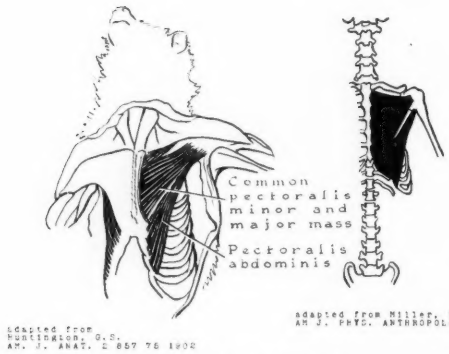


Fig. 3. Pectoral musculature in the lemur (from the original of Fig. 4, Plate 3, G. S. Huntington, *Am J. Anat.*, 1903, 2:157–175 and modified from Plate 7, by Ruth A. Miller, *Am. J. Phys. Anthrop.*, 1932, 17:53).

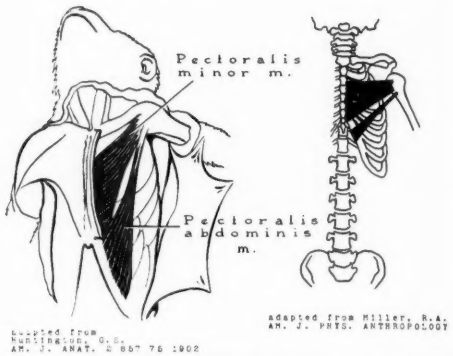


Fig. 4. Pectoral musculature in the old world monkey (kra) (from Fig. 5, Plate 4, by G. S. Huntington, *Am. J. Anat.*, 1903, 2:157–175, and modified from Plate 7, by Ruth A. Miller, *Am. J. Phys. Anthrop.*, 1932, 17:53).

end of the growth period. The fact that torsion continues until puberty is of interest in view of the prevalence of recurrent dislocations in young adults.

ANATOMIC FINDINGS

Seib dissected 1,000 shoulders on 500 cadavers and demonstrated that in 1 per cent of the shoulders, the pectoralis minor actually inserted onto the humerus; this probably represents an atavism, since this is the point of insertion in earlier species. Furthermore, in 15 per cent of the shoulders, there was some deviation from the normal coracoid insertion in his series. Most authorities believe that the coracohumeral ligament represents the vestigial remains of the insertion of the pectoralis minor onto the humerus.

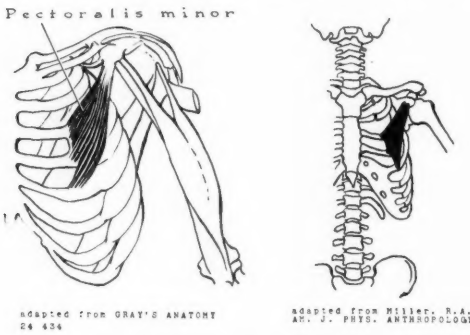


Fig. 5. Pectoral musculature in man (from Fig. 394, Gray's Anatomy, 25th ed., edited by Charles Mayo Goss, courtesy of Lea & Febiger, and modified from Plate 7, by Ruth A. Miller, *Am. J. Phys. Anthrop.*, 1932, 17:53).

DYNAMICS OF THE SHOULDER

As a dynamic apparatus, the shoulder obeys the laws of rotary motion. As the arm moves in internal or external rotation, there are two forces which must be controlled, the centrifugal and the rotatory swing. The centrifugal force *X* (Fig. 9) represents the tendency of a revolving body to leave its center of rotation. The rotatory swing *Y*, the more important, is the tendency of a rotating body to continue in motion in a tangential direction. Muscles exert both a rotatory and a stabilizing force. The rotatory force depends upon the angle of insertion of muscles. As the angle increases, the rotatory potential increases (Fig. 10).

The evolutionary changes described (humeral torsion, facing of the glenoid) have made it more difficult to control the forces in the extremes of external rotation. These developmental changes have given the short external rotators a comparative increase in their angle of application; that is, their rotatory potential has been increased (Fig. 10). The process of turning of the humeral head in the human embryo has already been illustrated (Fig. 8). The increased difficulty in controlling the centrifugal force *X* and the rotatory swing *Y* is evident in the comparison (Fig. 11) of the position of the shoulder in the resting position with that in external rotation.

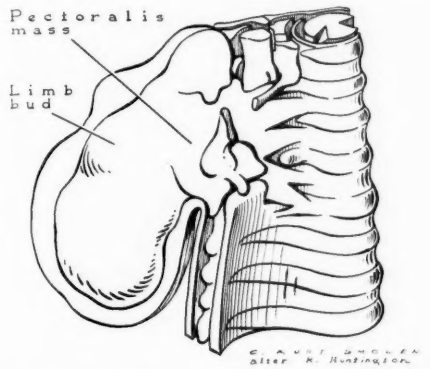


Fig. 6. Nine millimeter human embryo illustrating pectoral muscle mass (redrawn from Fig. 8, by Warren Harmon Lewis, *Am. J. Anat.*, 1902, 1:157).

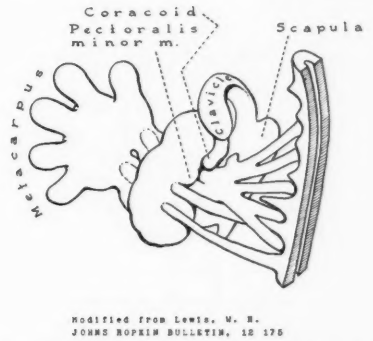


Fig. 7. Human embryo (11 mm.) showing bulging of pectoralis minor toward coracoid (from Fig. 4 by W. H. Lewis, *Bull. Johns Hopkins Hosp.*, 1901, 12:175).

COMMENT

From the study of the evolutionary and embryologic development of the shoulder and of the dynamics involved in its movements, the following significant facts emerge to account for the relative instability, but greater mobility, of the shoulder joint in man, as compared to his remote predecessors (Table I). A greater rotatory potential of the human shoulder has resulted from a change in thoracic configuration. There has been a comparative increase in the rotary potential of the short external rotators. There has been an increase in the short rotators as the result of greater scapular purchase. The pectoralis minor has been lost as an active internal rotator. There has been a change of the point of insertion of the pectoralis minor from the humerus to the coracoid. An interesting corollary of the fact that the point of insertion of the pectoralis minor has moved is that anatomic studies on human cadavers have shown that in a significant number of cases (15 per cent) the coracoid insertion deviates from the normal, and that in 1 per cent, the pectoralis minor is actually inserted onto the humerus, as in more primitive species.

All these factors re-emphasize the practical importance of controlling external rotation. It is in the position of external rotation and

TABLE I.—MYOLOGY OF THE SHOULDER

	Pectoralis major	Supraspinatus	Infraspinatus subscapularis	Pectoralis minor
Lemur	Common insertion with pectoralis minor in "common pectoral mass"	Origin from supraspinatus fossa and inserted upon greater tuberosity of humerus	Increase in width of earlier mammals	Insertion with pectoralis major in "common pectoral mass"
Old world monkey (kra)	Separate insertion onto the humerus	More narrow	Muscles have greater width	Separate muscle inserts onto the humerus
Man	Separate insertions	Most narrow	Approaches greatest width	Migration cephalad of insertion to coracoid

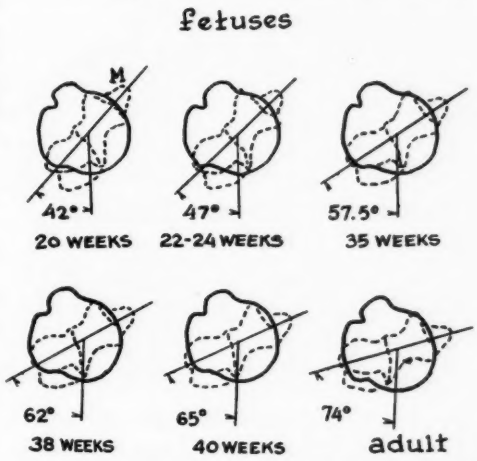


Fig. 8. Development of the humerus showing increasing torsion of the head (redrawn from the original of Fig. 10, by Vernon E. Krahl, *Am. J. Anat.*, 1947, 80:308).

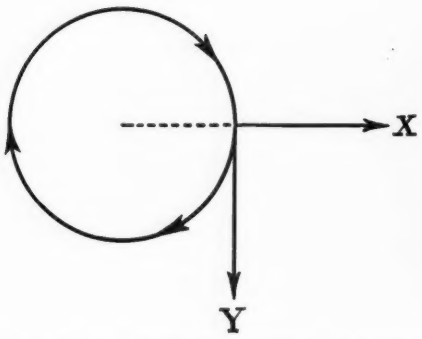


Fig. 9. Diagram illustrating rotatory swing Y and centrifugal force X, in rotary motion (from Fig. 1 (VI), p. 64, Arthur Steindler, ref. 18).

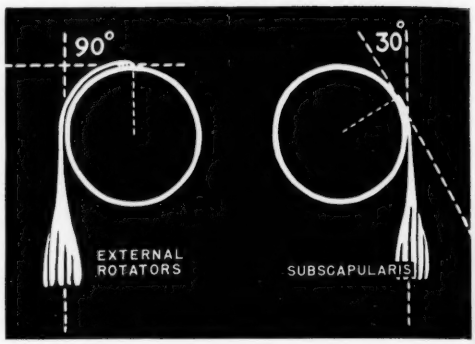


Fig. 10. Comparative increase of the rotatory potential of the external rotators owing to increase of the angle of insertion.

abduction that the majority of anterior dislocations of the shoulder occur. This is the position in which the shoulder joint has its least mechanical support anteriorly and if subjected to continuing strains beyond that of the muscle control, dislocation is apt to take place. This combination not infrequently happens in donning an overcoat or throwing the arm forcibly backward to catch a basketball or suddenly reaching upward for a hat.

The findings in our study offer a suggestion as to the etiology of the familial tendency observed in some cases of recurrent dislocation of the shoulder, of bilateral recurrent dislocations, and of recurrent dislocations in the presence of convulsive states. The developmental process which has increased the power of rotary motion in the shoulder has resulted in decreasing the stability of the joint. The relative increase in power of the external rotators, together with the torsion of the humerus and the changes in the facing of the glenoid, all play their part in the possibility and likelihood of a dislocation. These changes in development do not reach their ultimate until puberty, which may help to explain the frequency of recurrent anterior dislocation in young, muscular individuals. Any injury resulting in weakening of the structures which

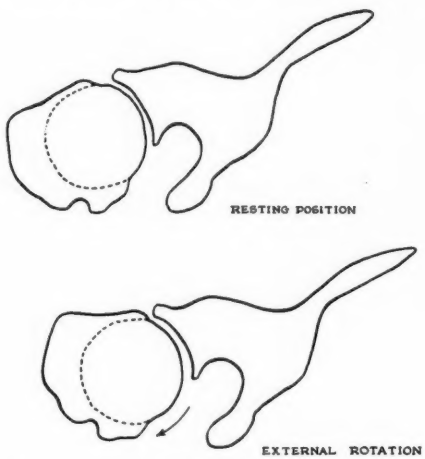


Fig. 11. Comparison of position of the shoulder in the resting position with that of external rotation; X indicates centrifugal force and Y the rotatory swing (see Fig. 9).

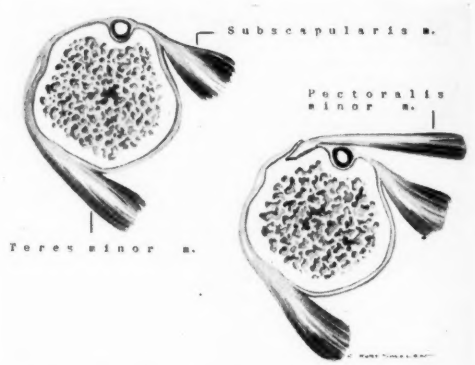


Fig. 12. Diagrammatic cross section demonstrating restoration of the insertion of the pectoralis minor onto the humerus.

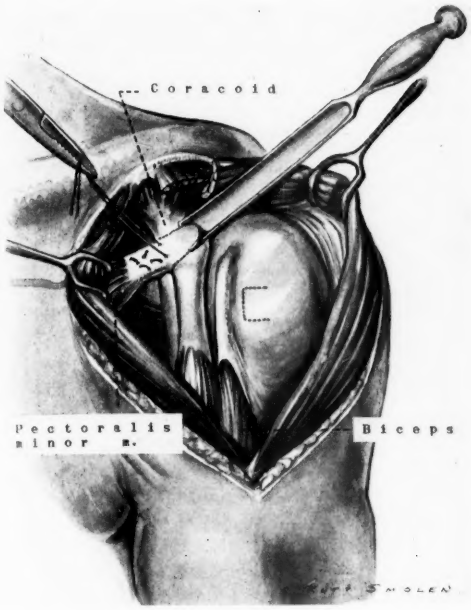


Fig. 13.

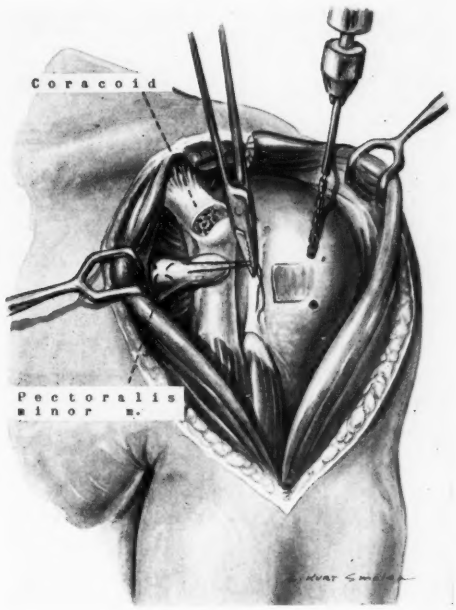


Fig. 14.

Figs. 13, 14, 15. Details of surgical technique.

limit external rotation may accentuate this inherent imbalance and result in recurrent dislocation. These same changes in development are compensated in later years by the fibrosis which is associated with the aging process.

SUGGESTED SURGICAL APPROACH

The object we had in mind when this investigation was initiated was to correlate the phylogenetic, ontogenetic, and dynamic aspects that might play a part in the development of a muscular imbalance influencing recurrent dislocations of the shoulder. However, as the study proceeded, the findings suggested that stability might be enhanced by restoration of the pectoralis minor as an active internal rotator.

It has been generally agreed by all who have directed special attention to this problem that any surgical procedure which controls external rotation is successful in the treatment of recurrent dislocation of the shoulder. As Watson-Jones has stated: "Whether there is a

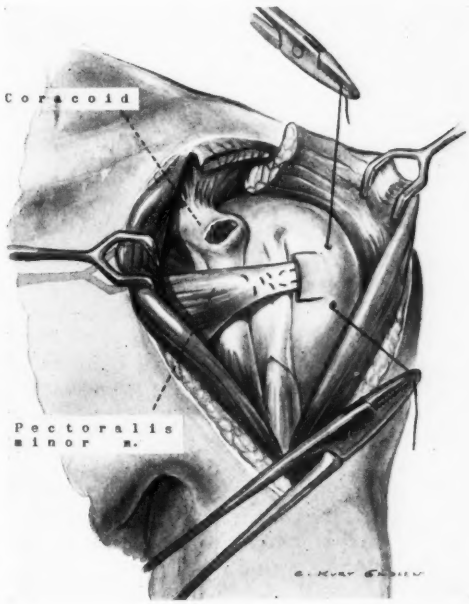
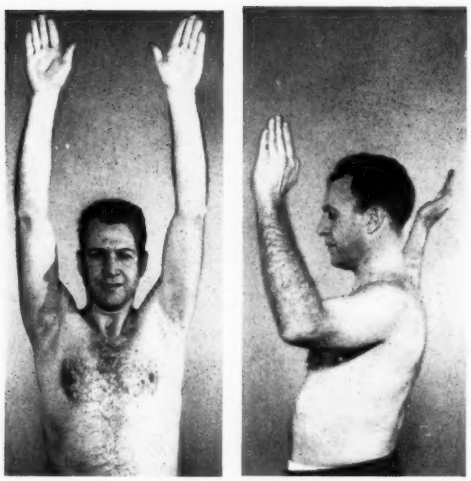


Fig. 15.



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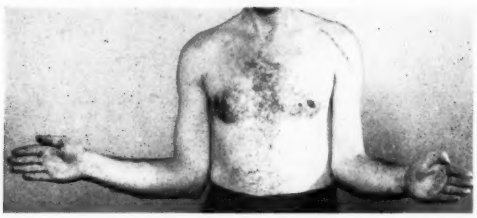

b

Fig. 16. Man, aged 32 years, 1 year after operation for recurrent dislocation of the shoulder: a, Full overhead extension of arm; b, 30 degree limitation of external rotation, with the arms at the side; c, profile view of 30 degree limitation of external rotation.

capsular defect or not, the extremes of external rotation must be controlled." Various surgical techniques accomplish this. For example, the Bankart (2–4) operation limits external rotation by tightening the capsule; the Putti-Platt (15) technique controls the external rotation by plication of the capsule and the subscapularis; the Gallie operation limits external rotation by the formation of new ligaments; the Magnuson-Stack (13) procedure controls external rotation by moving the insertion of the subscapularis, which we believe increases its rotatory potential. DePalma has adopted a modified Magnuson-Stack procedure.

Our study on the development of the shoulder suggested still another possibility, namely,

TABLE II.—PHYLOGENETIC AND DYNAMIC STUDY OF THE SHOULDER

1. Loss of pectoralis minor as an internal rotator.
2. Torsion of the humeral head resulting in inward facing.
3. Outward facing of the glenoid.
4. Medial migration of the bicipital groove, thereby decreasing the rotatory potential of the subscapularis.
5. An increased measurement in the coronal plane of the chest, thus facilitating rotary motion.
6. An increase in the rotatory power of the short rotators of the shoulder owing to increased length of the scapula.
7. Comparative increase in the rotatory potential of the short external rotators.

to restore the pectoralis minor to its original position, as seen in the embryo and in evolutionary forms, and also in 1 per cent of adult shoulders (Fig. 12). The reasoning behind this is that an active muscle would behave according to Sherrington's law of reciprocal innervation and would exhibit tension in all phases of rotation. It should prove to be an active inhibitor of external rotation.

Consequently, the following operative procedure was devised: A deltopectoral incision is made. A portion of the deltoid may be dissected from the clavicle to facilitate exposure of the humerus. With the deltoid reflected, the insertion of the pectoralis minor is isolated (Fig. 13). An interlacing silk suture is placed in the tendinous portion. A small curved chisel is then used to remove a cortical flap from the coracoid along with the insertion of the muscle. It must be emphasized that the suture must be securely and accurately placed before removal of the cortical flap. The flap must be of sufficient size to preserve all the tendinous attachments. Unless these precautions are observed, the tendon may fray or be placed under undue strain.

A small trap door is made on the greater tuberosity just lateral to the bicipital groove (Fig. 14). The groove is made over the center of rotation so there will be no increased tension in abduction. Two drill holes are made just lateral to the trap door. With the arm in internal rotation, the pectoralis minor is then brought over to the humerus and placed in the trap door by fastening the silk suture securely (Fig. 15). Additional stabilizing sutures may be added. Our experience has shown that the tension of the transplanted muscle is comparable to its tension in its original position.

Testing of the transferred tendon demonstrates tightening of the muscle in external rotation, but there is no increase in tension in any other position.

Postoperative care consists of fixing the arm to the body for 4 weeks in a modified Velpeau bandage. Further immobilization is carried out in a sling, and the patient is taught exercises which strengthen the internal rotators. Gradually normal activity is resumed. Complete rehabilitation is established to 12 weeks after operation.

We have carried out this procedure in 16 cases of recurrent dislocation of the shoulder. In additional 25 patients have been operated upon by various colleagues who have kindly furnished us with their records. The early results following this procedure have been most gratifying. The muscle transplant has resulted in a restriction of external rotation of about 30 degrees. This limitation of external rotation has been maintained and in no way interferes with everyday function of the shoulder (Fig. 16). Some of these patients have returned to active athletics and full military duty. The study, however, is still in a preliminary phase, since all the operations according to this technique have been performed in the past 2 years.

The surgical evidence accumulated to date indicates that extremes of external rotation must be controlled to achieve satisfactory therapeutic results in cases of recurrent dislocation of the shoulder. If the control of external rotation is the only factor to consider in designing a surgical procedure to correct this condition, then one must agree with DePalma that "the simplest procedure which will bring about restriction of the arc of external rotation will eventually be uniformly adopted." Certainly control of the extremes of external rotation is accomplished simply and easily by the operation we have suggested. From the phylogenetic and ontogenetic background of the pectoralis minor, it seems reasonable to suppose that changing its attachment from the coracoid to the humerus, where it is situated in more primitive primates, will restore the muscular balance lacking in these cases of recurrent anterior dislocation of the shoulder and thus provide satisfactory stability and good clinical results. Time alone will prove whether or not control of external rotation, of itself, gives sufficient stability in these cases to confirm or contradict the validity of this assumption.

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THE VALUE OF FRESH FROZEN SECTIONS

"WE shall have to wait for paraffins." So frequently have some surgeons heard this discouraging proclamation that they have given up making requests for fresh frozen sections. Others have been patiently led to believe that preliminary boiling of fresh tissue in formaldehyde is necessary for best results and that the 15 to 20 minutes required for the staining of such preparations with hematoxylin and eosin pass quickly. Still others suspect that their fresh surgical specimens are diagnosed grossly by the experts in the laboratory, with sound effects occasionally provided in the form of sporadic blasts on the freezing microtome.

Such hocus-pocus is not the subject of these comments. Moreover, the author is not alone in affirming that, with the exception of cortical bone and teeth, there is no solid tissue which cannot be cut, stained, and mounted for microscopic examination under an oil immersion lens—all within 2 minutes of the time of its surgical removal from the human body. No less is it a fact that with the use of polychrome methylene blue stain, there are no endogenous solid tissue elements which are not as clearly demonstrated as with the routine hematoxylin and eosin preparations made after 24 hours of fixation. Indeed, many elements, such as elastic tissue, glial fibrils, amyloid, hyalin, colloid, mucus and so forth, are almost as well demonstrated with polychrome methylene blue as with time-consuming specific stains for these substances.

It is axiomatic that proper surgical treatment is predicated upon correct diagnosis. Experienced surgeons are excellent diagnosticians of gross changes but gross appearance can deceive. Thus, the very obviously metastatic pleural nodules were secondary indeed —but derived from a mineral oil granuloma of the lung, and the elucidation of this diagnosis took 72 seconds. The manifestly tuberculous peritonitis was quickly proved, in 2 cases, to be granulomatous—but derived from ruptured ovarian dermoids; oophorectomy and peritoneal "scouring" was the operation indicated.

If pathologists are willing, however grudgingly, to concede that material from these and other conditions can be prepared and examined expeditiously, the benefits to be derived from such examinations performed *routinely* assume a role of importance *to the patient.* If, on the other hand, the pathologist under such pressure of responsibility is confronted with an impasse in diagnosis, "nothing achieved" is balanced by "nothing lost."

There are certain typical questions the answers to which, as afforded by the employment of fresh frozen sections, may save the patient time and expense in the hospital, reduce the number of operations necessary, or alter the type of operation which is performed.

1. *Is a particular inflammatory reaction specific or nonspecific?* An example of this problem is the patient with destructive arthritis. Grossly, at operation, the ragged, thickened synovial membrane resembles that of advanced rheumatoid arthritis, and arthroplasty is contemplated. But fresh frozen sections disclose an unmistakable granulomatous reaction with necrosis, and a tuberculous, brucellar, or other specific etiologic basis is suggested. As a result of this finding the operative procedure is modified, the operating room and the freezing microtome "decontaminated," and the special avenues of bacteriologic investigation narrowed.

2. *Is the lesion a neoplasm or an inflammatory condition?* This problem is illustrated by the middle-aged patient whose thoracic roentgenograms supported the clinical impression of dumbbell neurofibroma with large mediastinal and small spinal components. Fortunately, it was decided first to treat surgically that portion of the neoplasm that was compressing the spinal cord. At the time of laminectomy the "tumor" was yellow, friable, and well encapsulated—a neurofibroma, in other words. Biopsy, however, immediately established the diagnosis of a granuloma containing small, rounded, doubly contoured, nonsporulating organisms which were typical of Blastomyces dermatitidis. It is not unfair to assume that staying the hand of the surgeon saved the life of the patient in this instance. Subsequent therapy with potassium iodide and roentgen rays gave excellent results.

A second example is the dilemma of the surgeon when he palpates an indurated pancreatic head and is faced with the decision of performing or not performing a Whipple operation or some other major procedure. Few surgeons who operate for conditions within the upper portion of the abdomen have not faced this problem of deciding the issue of cancer versus pancreatitis. The Silverman needle can here be employed to secure multiple specimens for microscopic examination which can be carried out in almost as short a period of time as that required for removal of the specimens. A comparable procedure or one employing the principle of excision biopsy can be utilized to determine the nature of small nodules secured from the surface of the liver or elsewhere when the question is that of metastatic carcinoma versus healed tuberculosis, sarcoid, angioma, cholangioma, and so forth.

3. *Is the lesion a benign or a malignant neoplasm?* The majority of "special requests" for emergency frozen sections are perhaps made for the purpose of deciding this diagnostic issue in the case of lumps in the breast, and the problem should achieve solution in 99 per cent of cases when the rapid method of diagnosis is used. Even such a condition as plasma cell mastitis, the greatest masquerader of mammary cancer, is readily recognized microscopically although to the unwary it may mimic tuberculosis. Sclerosing adenosis, fetal fibroadenoma, Schimmelbusch's disease, Paget's disease, papillomatosis and so forth are lesions which, in the opinion of the author, are no more debatable when studied in fresh sections than they are in the average fixed tissue preparation. It is in the field of mammary pathology, then, that fresh frozen sections find their most widespread use, and in perhaps 20 per cent of cases the operative procedure

is altered as a result of what the surgical pathologist discovers. Comparable claims could be made in connection with cutaneous tumors, soft tissue tumors, bone tumors, brain tumors, gastrointestinal growths, and so forth, all of which lend themselves to this type of examination. One need but recall the infiltrating and obstructing propensities of sigmoidal endometriomas to realize how deceptive the gross appearance of some of these lesions may sometimes be.

4. Is the manifestly malignant tumor primary or metastatic? This question arises in a number of situations but the method of solution can readily be exemplified by the case of the young woman whose complaint of pelvic pain was apparently caused by the presence of a lower abdominal tumor. At operation the finding of signet-ring cells in a firm encapsulated ovarian neoplasm directed the surgeon's attention to an examination of the stomach where the small and clinically silent primary growth was located. Another example is that of two large cerebral tumors that were similarly diagnosed as metastatic seminomas arising from extremely small growths in the testicle.

5. Has the zone of surgical excision included the entire growth? In the treatment of basal cell carcinomas, epitheliomas associated with leucoplakia, and so forth, it is often difficult for the surgeon to delineate the extent of the tumor. Examining the edges of surgically excised tissues for evidence of residual carcinoma is a particularly useful function of the rapid freezing technique. The findings become especially important when, as in the case of large cutaneous defects, plastic reconstructive procedures are contemplated. Nobody likes to sew "cancer to cancer" even for cosmetic results.

Microscopic spread of malignant processes by means other than direct extension may be similarly determined. This includes blood and lymph vascular permeation, involvement of nodes, peritoneal seeding in the case of gastrointestinal new growths, and so forth. Many sections may be examined over a short period of time. Moreover, in each of the afore-mentioned circumstances, only those blocks containing positive or important evidence need be preserved for permanent record. Considerable time and effort are thereby saved.

6. Is the biopsy specimen representative? The bitterest holdout against the method will admit that even poorly prepared fresh frozen sections are adequate for purposes of deciding whether the excised tissue is alive or dead, and therefore diagnosable or not diagnosable by his or her routine tactics of delay and deferment. If the tissue proves unsatisfactory, the surgeon proceeds to remove other and perhaps more representative samples. Such fresh tissue "screening" is sometimes performed in the case of lymph nodes, synovial membranes, and muscle. With perseverance in the attempt to answer the question presented, the diagnosis as well as the adequacy of the specimen sometimes is simultaneously decided.

The foregoing remarks concern the use of a technically difficult method of examination which finds its natural role in hospitals with heavy surgical schedules. Its ends cannot be successfully achieved by the pathologist who, overtaxed with other responsibilities, is forced to defer performance of important duties to answer desultory summonses to the operating room. His laboratory may be three blocks or six floors from the surgical theater and his technician may have had woefully inadequate experience with the rapid freezing method of preparing sections. For such a pathologist to say that in his experience frozen sections are useless or even

misleading is perhaps a statement of fact. However, he is encompassing altogether too much territory if he affirms that definitive diagnosis cannot be made in a high percentage of cases when the method is used under ideal circumstances such as do not exist for his patient. MALCOLM B. DOCKERTY.

SIGNIFICANCE OF THE URINE EXAMINATION

ONE can believe that the English gentleman, Richard Bright, who first correlated proteinuria and anasarca with the pathologic demonstration of diseased kidneys, treated his subject gingerly, as befitted the fastidiousness of a man of his origin. Otherwise, it would be difficult to understand why Bright, who observed the existence of a urinary deposit at a time when the microscope was widely used in the rapid development of scientific pathology, made no attempt to study the minute structure of the deposit he observed. A similar fastidiousness must have led to the invention of the "sink test" by some unknown intern or technician. When one considers the casual way in which urine is ordinarily collected and prepared for examination, the disinclination of a physician personally to examine the urine can readily be understood.

The most important factor in a proper examination of urine is the technique for collection. When urine is collected in an unclean container and permitted to stand for hours at room temperature before the examination is performed, little significance can be attached to the observations. The specimen is overgrown with bacteria, the specimen has become alkaline from the action of urea-splitting organisms, casts are dissolved, and erythrocytes are lysed. For ordinary screening purposes a freshly voided (or catheterized, when indicated) specimen should be collected in a clean container and examined promptly, within the hour. For more thorough examinations, an overnight, timed specimen should be obtained in a clean container, to which a few drops of formalin should be added to inhibit bacterial growth.

Much information can be obtained from the fresh urine specimen, preferably collected under conditions of restricted fluid consumption, if the patient is known not to be azotemic. Such a specimen should be virtually free of bacteria, when the centrifuged sediment is examined. The color should be clear amber, or yellow, and the odor should not be unpleasant. If fluid consumption is withheld after dinner, the specific gravity of a morning specimen will usually exceed 1.020, and this is satisfactory evidence of normal tubular concentrating capacity, for practical purposes. The qualitative tests for sugar and protein should be negative and there should be no appreciable urinary sediment after centrifugation, other than a few leucocytes in the male or some squamous, vaginal epithelial cells in the female.

When bacteria are seen in the sediment of freshly voided urine it follows that they were present in the lower urinary tract. With stasis, such as that associated with prostatic obstruction, there may be a very low grade infection of the prostate gland or the bladder wall, sufficient to supply bacteria to grow in the warm, favorable culture medium with which the bladder is continually supplied, yet with insufficient tissue reaction to supply clumps of pus cells. When pus cells are seen, often agglutinated in clumps, the infection is associated with inflammation, and this may be the cause of such symptoms as dysuria, malaise, and fever. Infection of the lower tract alone does not ordinarily cause proteinuria. Serum contains 60 to 70 milligrams of pro-

tein per milliliter, and several milliliters may be exuded, diluted in the daily volume of urine, without production of a perceptible proteinuria. Rarely, exudative cystitis will confuse the issue. However, in general, proteinuria with bacteruria and pyuria may be taken to indicate renal disease.

Proteinuria may result from an abnormality of serum protein formation, such as that found in patients with multiple myeloma. Bence-Jones' protein has an abnormally small molecular size, compared to serum proteins, and consequently can slip through a normal glomerular membrane. Other abnormal proteins, such as hemoglobin formed by intravascular hemolysis, may also permeate the normal membrane. Under certain conditions, normal serum protein itself may permeate the glomerular membrane, as in patients who have received massive doses of serum albumin by intravenous administration. But, by and large, proteinuria is the result of renal disease, and the degree of proteinuria is often characteristic of the specific pathologic process. Thus, in renal infections the rate of proteinuria rarely exceeds 5.0 grams per day, while in degenerative tubular disease (e.g. nephrotic syndrome, acute renal ischemia syndrome, toxic nephrosis) the rate of proteinuria may exceed 15.0 grams per day. For these reasons, determination of the exact rate of protein excretion in a timed and measured urine specimen may have valuable diagnostic significance.

Hematuria is often of important diagnostic significance to the surgeon, since upon the discovery of hematuria may hinge the decision to operate. The common significance, which every student learns, is that hematuria associated with right-sided abdominal pain probably signifies a urinary calculus rather than acute appendicitis. It is important for the surgeon to remember that the hematuria of acute nephritis is usually gross, and the blood acquires the coffee-color of acid hematin before it is voided. Painless hematuria, without other symptoms, especially in the patients who are past middle age, may signify renal tumor. It is often forgotten that hypertension may cause a "renal epistaxis," just as nasal epistaxis is often caused. The occurrence of "renal epistaxis" is most frequent in patients who are also uremic, with the bleeding tendency that usually accompanies uremia. In young patients, hematuria may result from purpura of the Henoch-Schoenlein type. In such a case it is critically important to avoid instrumentation, since the trauma of ureteral catheterization may occlude the ureters and cause acute anuria.

Casts have a special significance in that they are formed within the tubules of the renal parenchyma. Thus, materials which are included in the substance of a cast must have been present in the kidney itself. For example, casts which include erythrocytes indicate that the blood originated within the renal substance and could not have been added during the passage through the lower tract, a conclusion which cannot be based upon the simple observation of hematuria. The same inferences may be applied to cells which are included in the body of a cast. Such cells must be derived from the tubular epithelium and indicate death and desquamation of the tubular lining. When such cells contain fat globules, they further indicate that the tubular epithelium had undergone fatty degeneration before desquamation, and this observation is pathognomonic of the specific pathologic process responsible.

Cellular casts are formed by the conglutination of desquamated, dying tubular epithelial cells. After the cast is formed, disintegration of the cells continues, first with the loss of cell boundaries and formation of large granules,

then with the formation of small granules, and finally with the fusion of all constituents into a waxy mass of high refractive index. The predominant nature of casts observed in the urinary sediment serves as a rough indicator of renal functional status. With diminished flow of urine through each nephron, local stasis occurs and the predominant cast type reflects the increased time for disintegration. In addition, casts are not ordinarily formed within the large collecting tubules. With severely impaired renal function the whole group of nephrons which empty into one collecting tubule may be affected. With this circumstance, urine flow through the collecting tubule may be reduced and casts will form with the large diameter characteristic of such large tubules. These casts, usually called broad casts, are also called "renal failure casts" since they are almost invariably associated with azotemia.

Miscellaneous other objects may be found in the urinary sediment, and it is important to know when objects should be ignored. Such judgment may only be gained by experience. Rotifers from fresh water used to rinse the container may be mistaken for parasites, which, with the exception of Trichomonas vaginalis, are rarely found in the urine. Motile sperm, occasionally found in male urine, may be mistaken for bacteria. The significance of crystals has been overemphasized by early pathologists, and, while crystals may usually be identified from their appearance, the knowledge gained is not ordinarily of clinical value. Chemical tests for a wide variety of unusual substances may be performed, and these are of diagnostic value in rare cases, such as pentosuria, cystinuria, and porphyrinuria.

Even a critical and observant *look* at the urine may provide valuable clinical information. Bacteruria and pyuria are often obvious at a glance. Impaired renal function may be revealed by the pale, dilute appearance of the morning specimen. Jaundice may first be evident in the urine, not by the typical yellow foam produced by bilirubin, but from the odd, dark, brownish-yellow color associated with urobilinogen and urobilin. Hematuria may not be evident at the first glance, but, since erythrocytes sediment more rapidly than other cells and casts, a red tip may often be seen under the button of centrifuged sediment, in the tube.

In recent years Papanicolaou has emphasized the value of information obtained from examination of desquamated material from the surface of internal organs. Recently the Papanicolaou technique has been applied to the urinary sediment for the detection of desquamated malignant cells from the upper urinary tract. It is important to remember that a study of the urine and its sediment offers a direct avenue for approaching the condition of the kidney, not only for the detection of malignant growths, but for direct information concerning the pathologic process in renal diseases. The chemical composition of urine further affords information concerning the kidney, and also concerning some aspects of general metabolism, in some ways reflecting its initial origin from the plasma. Few organs offer such ready and convenient access to the thoughtful clinician.

RICHARD W. LIPPMAN.

THE SURGEON'S LIBRARY

THE BOOK SHELF

B. J. D. IRWIN, THE FIGHTING DOCTOR

A Biographical Sketch

CLAUDE C. COLEMAN, M.D., F.A.C.S., Richmond, Virginia

ARIZONA stands alone in the number of doctors who practiced medicine while taking an active part in military expeditions in the latter part of the 19th century. Among these fighting doctors were B. J. D. Irwin, Leonard Wood, and G. E. Goodfellow. Walter Reed was an outstanding doctor in Arizona during this period, but took no part in the Indian fighting. Dr. Goodfellow was never a member of the military forces but worked with them on many occasions and because of his knowledge of the country his services were invaluable in the campaigns against the Apaches.

The subject of this sketch, Dr. B. J. D. Irwin, was born in County Roscommon, Ireland, in 1830 of Scotch-Irish descent. He came to this country as a boy and later studied medicine at the Castleton (Vermont) Medical School and the New York Medical College, graduating from the latter institution in 1852. He became one of the outstanding surgeons of America. His experience in the treatment of wounds was wide due largely to his service in the campaigns against the Indians, in some of which he commanded his detachment in addition to doing the necessary surgery.

Dr. Irwin showed a great proficiency in the use of firearms having on one occasion, according to his own statement, killed 175 birds with one shot and wounded about 25 others (2). On another occasion he killed two antelope with one shot of a rifle which passed through the heart of one and into the liver of the other.

He was always interested in the campaigns against the Indians, chiefly Apaches, who were then causing much trouble to the settlers of Southern Arizona and marauding at will. Dr. Irwin received for gallantry in action against the Apaches at Apache Pass the first Congressional Medal of Honor. Here he commanded a detachment while doing the necessary surgery (4). This medal was later received by Captain (later Major General) Wood as well as by Walter Reed, both of whom had conspicuous service in Arizona.

After a few years in southern Arizona Dr. Irwin came East to take part in the War Between the States and was made Inspector of the Army of the Cumberland. While holding this position he took part in the Battle of Shiloh (Tennessee, 1862) where he devised the first tent field hospital. This was a valuable forward step in the treatment of the wounded. This type of hospital was soon used by other armies and is now pretty well in general use all over the world.

General Irwin's medical interest lay not in battle wounds alone. He is also known for his treatment of rattlesnake bites with Euphorbia prostrata, a weed which grows in Arizona and other places, and is very easily applied in treatment of victims of snakebite (3).

Beginning as a Lieutenant in the Medical Corps of the Army, Dr. Irwin rose to the grade of Brigadier General and was retired at the age of 64, as required by law. In the meantime he had become assistant to the Surgeon General. He was a man of good mind and with more facilities would indeed have reached a very high position among the surgeons of our country. It seems that little attention has been paid to the accomplishments of the surgeons of southern Arizona during the latter part of the 19th century. This is probably due to the fact that Arizona was an isolated place and communications with the states and outside world were very difficult. It was very hard to get to Arizona and much harder to get away. The people of the East knew practically nothing about Arizona and their attitude toward it may be expressed by what took place at a celebration in Tucson in 1880. In this year the Southern Pacific Railway came to Arizona and reached Tucson. The Arizonans liked to celebrate and they would

use any excuse for a celebration which, in those days and even later, was always a very hilarious occasion. In 1880 the mayor of Tucson decided to give a big party to celebrate the arrival of the railway to his town. The celebration took the form of telegrams being read that were received from prominent people, such as the president of the United States, U.S. senators, and governors, congratulating the mayor upon the progress of Tucson. After each congratulatory telegram was read the crowd would go to the bar and take a drink. After a time the telegrams decreased in number and when one was read from Queen Victoria they changed from drinking whiskey to champagne. Among the guests there was a crowd of joy-makers from Tombstone. These guests, fortified by liquor, were somewhat disgruntled because Tucson got the railway and the party and they did not, so knowing that a telegram had been sent to the Pope they went out and fixed up the following reply: "With great apostolic joy His Holiness congratulates the Mayor of Tucson upon the progress of his city. In order that he may keep in contact with the further progress of this progressive city His Holiness would like to know 'Where in Hell is Tucson anyway?' Signed, Antonelli, Papal Secretary." The East would probably have endorsed the Pope's question wholeheartedly.

The United States Government had become very much concerned over doing anything about Arizona. The Apaches would raid the settlements, often kill the inhabitants by cutting their throats and drive off the stock. It was almost impossible to capture them. If it had not been for the discovery of minerals, such as copper and silver, it is not unlikely that the United States would have abandoned Arizona to the Apaches. It was said that General Sherman desired to do so. He had been sent on one occasion during the Apache difficulties to Arizona to study the situation and to make suggestions to the President for the control of the Indians. General Sherman was very much of a realist. The Hell that he made the war in the East was nothing in violence and brutality compared to the Apache warfare. He was said to have summarized his conclusions at this time about Arizona in the following statement: "We fought Mexico a big war to get this country. Let's fight another one to make them take it back!" When told by some of the citizens who were very anxious to get the soldiers to protect the settlers that Arizona was "really" not a bad country—it only lacked water and society, Sherman was said to have responded, "That's all Hell lacks."

General B. J. D. Irwin

Of course, when the soldiers of the United States Army were withdrawn from Arizona to go East to fight against the South the Apaches thought they had run them away and there was great rejoicing and much marauding. It got so bad that the South Arizonans were afraid to live on the farms and moved into the towns and missions. The discovery of silver and copper in large quantities caused the adventurous people of the West to keep up the fight against the Apaches. In some ways the cruelty of the settlers was often equal to that of the Apaches. The scalping of the victims of the Indian warfare was not uncommon although the Apaches were not inclined to scalping. They preferred as a terminal brutality the crushing of the victim's head with a rock. When the United States Government started paying a bounty for Indian scalps it then became a commercial undertaking.

This brief reference to Indians and Indian warfare appears to be permissible in this sketch of Dr. Irwin inasmuch as his practice of medicine in the army was always done under conditions of great risk. On one occasion he rode on horseback 115 miles in 24 hours and operated upon one of the soldiers who had been so badly injured that the

amputation of his left arm at the shoulder was necessary. The man was in bad shape from loss of blood. After the operation Dr. Irwin took him to a fort some 60 miles away in an ordinary wagon over a rough road through a country infested by hostile Indians. The patient made a good recovery.

Dr. Irwin died in 1917 at the age of 87 years. He was a man of splendid physical as well as mental equipment, and contributed much to the colonization of the Southwest. He had held a number of positions of honor and importance. He was a past president of the Order of Military Surgeons of the United States and during that term of office (1893–1894) he was sent to Rome as a delegate of the United States Government to the Eleventh International Medical Conference. His remains lie at West Point.

In appearance Dr. Irwin was a tall, erect, fine looking man, whose physical equipment was equal to the demands made upon it by the hardships of Indian warfare. He was said to be an expert linguist in that he could speak five different languages (1).

A monument has been erected at the battlefield of Shiloh with the following inscription: "First Tent Hospital ever erected in time of war was established by Captain B. J. D. Irwin, U. S. Medical Corps." This was a very important discovery—an outstanding achievement in a great man's career, making possible the early treatment of the wounded soldier. This is even more important now since the days of antiseptics, aseptic surgery, and antibiotic drugs.

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3. Idem. Notes on Euphorbia prostrata as an antidote to the poison of the rattlesnake. From the files of the Arizona Pioneers Historical Society.
4. Thompson, W. M. The fighting doctor. Southwest. M., May, 1935.

REVIEWS OF NEW BOOKS

THE purpose of *A Color Atlas of Morphologic Hematology*[1] is to furnish a guide for reference in the study of films of peripheral blood dyed with Wright's stain. It is not a manual of techniques. For these one is referred to *A Syllabus of Laboratory Examinations in Clinical Diagnosis* which is a companion book to this atlas.

The book is divided into 2 parts. The first section deals mainly with the general characteristics of the formed elements of the blood as dyed with Wright's stain. The nucleus, cytoplasm, and granules of the erythrocytic series and the different leucocytes are described in detail according to the maturation of the cells in each series. There are excellent pen and ink diagrams of the maturation of each type of cell, as well as a colored plate of the erythrocyte series and one of normal leucocytes. The second part of the book consists of case studies of various disorders of the blood. There are 12 colored plates representing the blood picture in different anemias, leucemias, and in infectious mononucleosis. Most of these are divided into 2 parts representing different stages of the disease. A description and interpretation is given of each picture as well as the laboratory data of the case.

The text is clearly written, though the small print detracts from the book. There are 74 pages with a bibliography of 193 articles. The colored plates and black and white diagrams are excellent. The book accomplishes its purpose and should be a great help to clinicians, clinical pathologists, technicians, and medical students; not only in the study of the morphology of blood cells, but also in the interpretation of the blood pictures characteristic of various diseases of the blood. OPAL E. HEPLER.

THE fifth edition of Kolmer, Spaulding, and Robinson's *Approved Laboratory Technic*[2] has been extensively revised, largely rewritten, and considerably enlarged by the inclusion of newer methods and new illustrations. This expansion includes 163 additional pages, 57 new figures, and 24 new tables.

It is impossible to list all of the new laboratory procedures included, but some of the more important are phase microscopy, blood volume, Papanicolaou cytodiagnosis of cancer, the V. D. R. L. method for quantitative estimation of spinal fluid protein, the *Rana pipiens* test for pregnancy, thymol turbidity, and flame photometry. The old colored plates of blood cells have been replaced by new pictures and the number increased. Newer methods for the titration of Rh antibodies have been added. Part 3 which includes bacteriologic, mycologic, and virologic methods has been greatly improved. A helpful addition is an appendix which includes conversion factors, weights and measures, milliequivalents and millimols, atomic weights, and four place logarithm tables.

The general character of the book has not been changed. The uniform method of presentation of each test—that is, the principle, the technique, and sources of error—make the book an excellent guide for technicians. The interpretation or significance of the results of the tests is only briefly discussed. This volume is, therefore, a companion book for *Clinical Diagnosis by Laboratory Methods* by the senior author. This new fifth edition continues the high standard of the previous editions and should be available to every clinical pathologist and laboratory technician as a laboratory guide and reference book. OPAL E. HEPLER.

THE author of *Kymographische Roentgendiagnostik zur Beurteilung des Herzens in Beispielen*[3] who was principally responsible for the adaptation of kymography to clinical cardiovascular infections, now reports on 25 years' experience with the procedure. As the name implies, the purpose of the method is to register the movement of the various segments of the cardiovascular contour in graphic form by way of stationary films and moving grid. The same points in the cardiac contour always appear in the slits in one and the same phase of movement. Kymograms made with the moving grid and the stationary films are sometimes preferable, especially by those without much experience. Cardiac kymography is a two dimensional registration of three dimensional movements.

The author submits his series of 40 cases with elaborate documentation, together with the kymographic evaluations as evidence of the value of the procedure. It is a method thus far little used, but for which it is anticipated that internists will find increasing indication. JAMES T. CASE.

THERE is a culmination of 18 years of intensive study in Edith L. Potter's *Pathology of the Fetus and the Newborn*.[4] The author bases her material upon information gained from the study of more than 6,000 autopsies performed on infants and fe-

[1] A COLOR ATLAS OF MORPHOLOGIC HEMATOLOGY WITH A GUIDE TO CLINICAL INTERPRETATION. By Geneva A. Daland, B.S. Edited by Thomas Hale Ham, M.D. Illustrations by Etta Piotti. Cambridge, Mass.: Harvard University Press, 1951.

[2] APPROVED LABORATORY TECHNIC. By John A. Kolmer, M.D., D.P.H., Sc.D., F.A.C.P., Earle H. Spaulding, Ph.D., and Howard W. Robinson, Ph.D. 5th ed. New York: Appleton-Century-Crofts, Inc., 1951.

[3] KYMOGRAPHISCHE ROENTGENDIAGNOSTIK ZUR BEURTEILUNG DES HERZENS IN BEISPIELEN. By Prof. Dr. Pleikart Stumpf. Stuttgart: Georg Thieme, 1951.

[4] PATHOLOGY OF THE FETUS AND THE NEWBORN. By Edith L. Potter, M.D., Ph.D. Chicago: The Year Book Publishers, Inc., 1952.

tuses at the Chicago Lying-in Hospital, on the study of tissues from an additional 3,000 autopsies performed by pathologists from the Chicago Department of Health, and on a review of 10,000 protocols of autopsies on live born infants under 1 year of age who die in other Chicago hospitals. Although emphasis is placed upon lesions of the fetus and newborn, there also is a consideration of conditions which affect the infant during the first 3 months of extrauterine life. The pathology of the embryo is not dealt with in detail.

The organization of the text is such that the reader first reviews the early development of the fetus and placenta. Subsequent chapters discuss the rate of antenatal growth, the normal and abnormal chorionic vesicle and placenta abortion, the causes of fetal and infant death, the postmortem examination of the fetus and newborn, prematurity, anoxia, birth trauma, infections, malformations, tumors, multiple pregnancies, and conjoined twins. Pathologic changes occurring in the various body systems are covered in detail in the remaining chapters. A comprehensive list of references, limited mostly to the English literature, is appended to each chapter.

Concise histopathologic details are presented with emphasis on the important characteristics of the lesions being discussed. Throughout the text the author has utilized her wealth of material to integrate the clinical background and the clinical application with the pathologic picture of the particular entity under consideration.

Dr. Potter's work is an outstanding addition to the medical literature. It constitutes a reference of inestimable value to the pathologist, the obstetrician, and the pediatrician. The 601 illustrations are uniformly good and are well reproduced. In addition, it is an excellent example of the medical photographer's, the engraver's and the bookmaker's art.

JOHN W. HUFFMAN.

WITH the publication earlier this year of the second volume of the twenty-first edition of Erich Lexer's celebrated *Lehrbuch der allgemeinen Chirurgie zum Gebrauche fuer Aerzte und Studierende*[1], German-reading students and surgeons have the opportunity of exploring a work that has been established as a classic in surgical literature since its first publication in 1904, and has remained so through 21 editions.

Although Lexer died in 1937, there is no discernible threat of mortality in this revision by Eduard Rehn of Freiburg University and his able staff of collaborators. Distinguished pupil of, and successor to Lexer, Professor Rehn, with his group, has brilliantly spanned the 20 years that separate the twentieth and twenty-first editions. Enormous achievement, both in surgery and medicine, has been made during these years. The task that Rehn and his associates accepted, when they set out to bring up to date a masterpiece, as Lexer's book is, and live up to its high standard, must have been difficult but also challenging. They are to be congratulated.

The first volume of this edition was published 3 years ago. With the appearance now of the second volume, a critical evaluation of the entire work is possible. Its title defines its scope and content. This is clear to the German reader, who thinks of *Lehrbuch der allgemeinen Chirurgie* as a textbook of the principles and practice of surgery. This is not so for the English reader, since *allgemeine* is frequently regarded as the counterpart of the word "general," which, when applied to surgery, means general surgery as contrasted with surgery of its specialties. The difficulties in rendering *allgemeine* as "general" required A. D. Bevan and Dean Lewis, in 1910, in their American edition of Lexer's *Lehrbuch* to adopt the subtitle, *A Presentation of the Scientific Principles upon Which the Practice of Modern Surgery Is Based.* This, then, is the kind of book that Lexer wrote.

Many changes have been made in the text and new chapters have been added. Among these valuable innovations is the chapter on therapy with biocatalysts (vitamins, hormones, and ferments) by Dr. Marquard, as well as the one on constitution in surgery by Professor v. Brandis. In the second volume few changes have been made in the chapter on mechanical injuries and reference to recent foreign literature dealing particularly with tendon, nerve, and vessel surgery is unfortunately missing. Other chapters have been thoroughly modernized, and these include vitally useful information on chemical and thermal injuries, shock, fat embolism, and surgical diseases of skin, mucous membranes, muscles and tendons, tendon sheaths and bursae, vessels, peripheral nerves, joints, and bones. Finally, as a grand finale, there is a chapter on the behavior, growth, and cause of tumors by the late Ludwig Aschoff's distinguished pupil and successor, Professor Büchner, and Professors Liebegott and Jung.

There are no outward, physical changes from the previous edition. The paper and binding, as well as the reproduction of the illustrations, many of them in color, are of the same high quality as before and reflect the traditional craftsmanship of the publisher.

In a sense, this is a fitting memorial to Erich Lexer. But even more important, through the efforts of Professor Rehn and his associates this work will continue to broaden and advance for medical students and surgeons who read the German language the knowledge of the scientific principles and practice in surgery. It is hoped that this modern revision will be translated into English.

HANS MAY.

A real contribution to the literature of thoracic surgery has been made by Dr. Hochberg in his book on the care of *The Thoracic Surgical Patient.*[2]

[1] LEHRBUCH DER ALLGEMEINEN CHIRURGIE; ZUM GEBRAUCHE FUER AERZTE UND STUDIERENDE. By Erich Lexer. New work by Prof. Dr. Eduard Rehn. Vol. 2, 21st ed. Stuttgart: Ferdinand Enke, 1952.

[2] THE THORACIC SURGICAL PATIENT; PREOPERATIVE, ANESTHETIC, AND POSTOPERATIVE CARE. By Lew A. Hochberg, M.D. Foreword by Frank B. Berry, M.D. New York: Grune & Stratton, 1952.

The best part of this book deals with the physiology involved in patients with thoracic injuries and diseases. This section alone makes his contribution worthwhile, and is particularly valuable since practically all of the recent books on thoracic surgery have omitted this important subject.

In a thorough manner he has considered every type of injury and disease affecting the chest, and while the main body of his work has to do with preoperative and postoperative care he has briefly, but accurately, sketched the diagnosis and operative management of these conditions. While the reviewer is in general agreement with Dr. Hochberg on all subjects he has discussed there are many details with which he is not in agreement. However these are not important enough to warrant discussion.

The author obviously has based the book on his experiences in a large thoracic surgical service in a large urban hospital. Because of this, his suggestions for the care of the patient presupposes the services to be found in such an institution. However, the surgeon without such facilities will find that he can follow Dr. Hochberg's directions by performing the procedures himself, or by training his available helpers to do so. This book is, therefore, of value to everyone interested in thoracic surgery and meets a real need. RICHARD H. MEADE.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

A HANDBOOK OF OPERATIVE SURGERY; SURGERY OF THE CHEST. By Julian Johnson, M.D., and Charles K. Kirby, M.D. Chicago: The Year Book Publishers, Inc., 1952.

A TEXT-BOOK OF PATHOLOGY. By E. T. Bell, M.D. Contributors: B. J. Clawson, M.D., and J. S. McCartney, M.D. 7th rev. enl. ed. Philadelphia: Lea & Febiger, 1952.

THE PRINCIPLES AND METHODS OF PHYSICAL DIAGNOSIS; CORRELATION OF PHYSICAL SIGNS WITH PHYSIOLOGIC AND PATHOLOGIC CHANGES IN DISEASE. By Simon S. Leopold, M.D. Philadelphia, London: W. B. Saunders Co., 1952.

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CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

ALTON OCHSNER, New Orleans, *President*
HAROLD L. FOSS, Danville, Pennsylvania, *President-Elect*

PRELIMINARY PROGRAM FOR THE 38TH CLINICAL CONGRESS, THE WALDORF-ASTORIA, NEW YORK, SEPTEMBER 22 THROUGH 26, 1952

THE most informative and intensive meeting in the history of the American College of Surgeons will unfold at the 38th annual Clinical Congress in New York on September 22 through 26. New sessions on subjects currently receiving professional and public interest, plus a greater number of meetings on vital subjects, promises a Clinical Congress of unusual scope and variety.

Innovations this year include a motion picture symposium on surgery of the heart and a separate three day program in gynecology and obstetrics. Specialties being increased are neurologic, thoracic, orthopedic, plastic, and urologic surgery. Scientific papers in the Forum on Fundamental Surgical Problems are being increased from 142 last year to 185 this year. Two additional postgraduate courses are being given. Ophthalmology and otolaryngology will have their own sessions on Tuesday, Wednesday, and Thursday.

This year's Congress will also have a larger number of hospitals participating than ever before, a total of 63. Other sessions throughout the week will include panel discussions, Ciné clinics, color television, medical motion picture films, symposia on cancer and on trauma, and scientific and technical exhibits.

POSTGRADUATE COURSES

Seven postgraduate courses will be given mornings, from 9:00 a.m. until 12 noon, Tuesday through Friday. Courses will be given at Lenox Hill, Mount Sinai, and Bellevue Hospitals and the Waldorf-Astoria, Belmont Plaza, and Biltmore Hotels. Tickets for the courses may be obtained at the clinic ticket desk beginning Sunday afternoon, September 21, after 3 p.m.

The fields in which courses will be given, and their Chairmen follow:

Cardiovascular Surgery. CLARENCE DENNIS, M.D., F.A.C.S., Music Room, The Biltmore.
Preoperative and Postoperative Care. JOHN H. MULHOLLAND, M.D., F.A.C.S., Le Perroquet Room, Waldorf-Astoria.
Fractures. ROBERT H. KENNEDY, M.D., F.A.C.S., Einhorn Auditorium, Lenox Hill Hospital.
Gynecology and Obstetrics. HOWARD C. TAYLOR, JR., M.D., F.A.C.S., Baroque Room, Belmont Plaza Hotel.
Nonmalignant Diseases of the Gastrointestinal Tract. RALPH COLP, M.D., F.A.C.S., Blumenthal Auditorium, Mount Sinai Hospital.
Trauma of the Central Nervous System. E. JEFFERSON BROWDER, M.D., F.A.C.S., Jansen Suite, Waldorf-Astoria.
Surgical Aspects of Pulmonary Disease. FRANK B. BERRY, M.D., F.A.C.S., Amphitheatre, Building C-1, Bellevue Hospital.

COLOR TELEVISION

Television programs, made possible by Smith, Kline & French Laboratories of Philadelphia, will emanate from Presbyterian Hospital and will be shown at Hotel Belmont Plaza Monday through Friday. Operations schedule for each day will be published in the *Daily Bulletin* of the Congress.

CINÉ CLINICS

As in previous years, the purpose of the Ciné clinics, sponsored by Davis & Geck, Inc., is to present a showing of surgical procedures representative of the surgery in the area of the Clinical Congress. Latest developments in the broad field of surgery, standard procedures in the various surgical specialties and the surgery of trauma will be covered in four Ciné clinic sessions. The selection of materials is the result of co-operative effort on the part of the Medical Motion Picture Committee, headed by Dr. Hilger

Perry Jenkins, and the Committee on Local Arrangements, headed by Dr. Frank Glenn. Dr. Henry Cave is chairman of the committee in charge of the presentation of these clinics. Clinics will be held Tuesday through Friday mornings, from 9:00 a.m. to 12:30 p.m. in the Grand Ballroom of The Waldorf-Astoria.

A tentative list of subjects and authors follows:

Acute Gallbladder. FRANK GLENN, M.D., F.A.C.S., New York.
Pyloric Stenosis. EDWARD J. DONOVAN, M.D., F.A.C.S., New York.
Tracheo-esophageal Fistula. GEORGE M. HUMPHREYS, M.D., F.A.C.S., New York.
Sphincter of Oddi. JOHN H. MULHOLLAND, M.D., F.A.C.S., New York.
Skin Grafting. HERBERT CONWAY, M.D., F.A.C.S., New York.
Internal Fixation of Fractures. HARRISON L. MCLAUGHLIN, M.D., F.A.C.S., New York.
Bleeding Peptic Ulcer. JOHN D. STEWART, M.D., F.A.C.S., Buffalo.
Regional Enteritis. L. KRAEER FERGUSON, M.D., F.A.C.S., Philadelphia.
Colectomy. G. GAVIN MILLER, M.D., F.A.C.S., Montreal.
Carcinoma of the Esophagus. SAMUEL P. HARBISON, M.D., F.A.C.S., Pittsburgh.
Surgery of the Liver. I. S. RAVDIN, M.D., F.A.C.S., Philadelphia.
Surgery for Acute Vascular Injury. JERE W. LORD, M.D., F.A.C.S., New York.
Intussusception. MARK M. RAVITCH, M.D., F.A.C.S., Baltimore.
Sympathectomy. I. RIDGEWAY TRIMBLE. M.D., Baltimore.
Surgery for Acute Chest Injuries. J. MAXWELL CHAMBERLAIN, M.D., F.A.C.S., New York.
Surgery for Acute Abdominal Injury. HOWARD A. PATTERSON, M.D., F.A.C.S., New York.
Hemipelvectomy. BRADLEY L. COLEY, M.D., F.A.C.S., New York.
Surgery for Acute Head Injury. BRONSON S. RAY, M.D., F.A.C.S., New York.
Surgical Débridement. ROBERT H. KENNEDY, M.D., F.A.C.S., New York.
Halsted Radical Mastectomy. WARFIELD M. FIROR, M.D., FA.C.S., Baltimore.

HOSPITAL CLINICS

A major feature of the Clinical Congress is the program of operative clinics and demonstrations being arranged by representatives from 63 hospitals in the New York area. Schedules of clinics, which will include general surgery, gynecology and obstetrics, fractures and other trauma, neurosurgery, orthopedic, thoracic, and genitourinary surgery, ophthalmology and otolaryngology will be published in the *Daily Bulletin* issues each day during Congress week. Tickets for these clinics may be procured at the clinic ticket desk beginning Sunday afternoon, September 21. Participating hospitals holding surgical clinics and their hospital representatives are as follows:

New York

Beekman–Downtown Hospital—LESTER BLUM
Bellevue Hospital
  First Division—AARON HIMMELSTEIN
  Second Division—ERNEST W. LAMPE
  Third Division—CARL SMITH
  Fourth Division—LOUIS R. SLATTERY
Beth David Hospital—LESTER BREIDENBACH
Beth Israel Hospital—HERBERT NEUMAN
Bronx Hospital—HERMAN B. SCHOENBERG
Flower and Fifth Avenue Hospitals—WALTER L. MERSHEIMER
Fordham Hospital—LOUIS MARTON
Francis Delafield Hospital—CUSHMAN D. HAAGENSEN
French Hospital—HOWARD A. PLANK
Goldwater Memorial Hospital
  Second Division—LOUIS CARP
  Third Division—BENJAMIN G. SHAFIROFF
Harlem Eye and Ear Hospital—EUGEN GRABSCHEID
Harlem Hospital—DAVID H. SMITH
Hospital for Joint Diseases—JOSEPH BUCHMAN
Hospital for Special Surgery—T. CAMPBELL THOMPSON
Knickerbocker Hospital—RUSSEL H. PATTERSON
Lenox Hill Hospital—HERBERT C. MAIER
Lincoln Hospital—WILLIAM H. CASSEBAUM
Lying-In Hospital—ARTHUR B. GREELEY
Manhattan Eye, Ear and Throat Hospital—R. TOWNLEY PATON
Memorial Center for Cancer and Allied Diseases—HENRY T. RANDALL
Montefiore Hospital for Chronic Diseases—ELLIOTT S. HURWITT
Morrisania City Hospital—RENATO J. AZZARI
Mount Sinai Hospital—ARTHUR S. W. TOUROFF
Neurological Institute—J. LAWRENCE POOL
New York City Hospital—PRESTON A. WADE
New York Eye and Ear Infirmary—RAYMOND E. MEEK
New York Hospital—S. W. MOORE
New York Orthopedic Hospital—ALEXANDER GARCIA
New York Polyclinic Medical School and Hospital—JOHN E. HAMMETT
Presbyterian Hospital—ROBERT H. E. ELLIOTT
Roosevelt Hospital—JAMES E. THOMPSON
St. Clare's Hospital—JAMES T. DANIELS
St. Luke's Hospital—PAUL CALHOUN MORTON
St. Vincent's Hospital—JOHN A. LAWLER, JR.
Sloane Hospital for Women—CHARLES M. STEER
U. S. Veterans Hospital (Kingsbridge)—PAUL K. SAUER
University Hospital—LOUIS R. SLATTERY
Woman's Hospital—ALBERT H. ALDRIDGE

Brooklyn

Beth-El Hospital—BENJAMIN KOGUT
Brooklyn Eye and Ear Hospital—CHARLES R. WEETH
Brooklyn Hospital—KENNETH H. MACGREGOR
Coney Island Hospital—J. EARL MILES
Cumberland Hospital—SAMUEL LUBIN
Greenpoint Hospital—SOLOMON SCHUSSHEIM
House of St. Giles the Cripple—ROBERT F. WARREN
Jewish Hospital—LOUIS BERGER
John E. Jennings Hospital—ALLISON J. VOSSELER
Kings County Hospital
  Kings County Division—JOHN F. RAYCROFT
  University Division—CLARENCE DENNIS
Long Island College Hospital—CHARLES B. JONES
Maimonides Hospital—ABRAHAM MANDELBERG
Methodist Hospital—C. DOUGLAS SAWYER
Norwegian Lutheran Deaconesses' Home and Hospital—HENRY A. MEHLDAU
St. Catherine's Hospital—JOHN A. MCCABE

St. Charles Hospital and Orthopedic Clinic—HERBERT C. FETT, JR.
St. John's Episcopal Hospital—MERRILL N. FOOTE
St. Mary's Hospital—ANDREW J. MCGOWAN
U. S. Veterans Administration Hospital—RALPH FRIEDLANDER
Wyckoff Heights Hospital—PHILIP A. ZOLLER

Jamaica, L. I.

Jamaica Hospital—JOHN HALLINAN
Mary Immaculate Hospital—VINCENT DEPAUL JUSTER
Queens General Hospital—CHESTER L. DAVIDSON

St. Albans

U. S. Naval Hospital—C. F. STOREY

Staten Island

St. Vincent's Hospital—JOHN K. LUCEY
Sea View Hospital—LOUIS R. DAVIDSON

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

The entire range of surgery benefits from the new ideas presented in the papers heard each year in the Forum on Fundamental Surgical Problems. The increased number of papers to be heard this year are in direct answer to the success and popularity of the Forum. The Surgical Forum Committee is headed by Dr. Owen H. Wangensteen.

AFTERNOON AND EVENING SESSIONS

There will be three general surgery evening sessions, Tuesday through Thursday. On Wednesday evening Dr. Sumner L. Koch of Chicago will give the annual Trauma Oration, speaking on "The Working Man's Hand." General surgery afternoon panel discussions will be held Monday through Wednesday afternoons. Separate sessions for ophthalmologists will be held Tuesday, Wednesday, and Thursday mornings and evenings. Otolaryngologists will have their meetings simultaneously. Special panel discussions in gynecology and obstetrics will be held Wednesday through Friday afternoons. Afternoon panel discussions in five surgical specialties will be held on Friday afternoon. The Symposium on Trauma will be held Tuesday at 2 p.m., with Dr. R. Arnold Griswold of Louisville presiding. The Symposium on Cancer will be held Wednesday afternoon with Dr. Edwin P. Lehman of Charlottesville presiding. Outlines of all programs appear on succeeding pages.

MOTION PICTURE SYMPOSIUM ON SURGERY OF THE HEART

On Tuesday evening at 8:30 there will be a motion picture symposium on surgery of the heart. The subject will be approached from several facets, including basic research, clinical investigation, and clinical surgery. Participants will provide their own narration during the showing of the films and there will be a question period following the motion pictures.

GRADUATE TRAINING SYMPOSIUM

Frederick A. Coller of Ann Arbor, Chairman, Graduate Training Committee, will preside over a session on "Residency Problems in Smaller Hospitals" on Thursday afternoon, 1:30 until 3 o'clock, in the Bowman Room of The Biltmore.

GENERAL ASSEMBLY AND CLINICOPATHOLOGY CONFERENCE

Dr. Alton Ochsner will preside at the opening General Assembly on Monday morning at 10 o'clock at which Dr. Frank Glenn, Chairman of the New York Committee on Arrangements, will greet the assembly. At the same meeting Dr. Gunnar Gundersen, Chairman of the Joint Commission on Hospital Accreditation, and Dr. Edwin L. Crosby, Director, will speak on the inauguration of the joint hospital accreditation program.

Dr. Ochsner will preside also over the Clinicopathology Conference at 11 a.m. Dr. Joseph E. Flynn, New York, will act as moderator at this meeting.

MEDICAL MOTION PICTURE PROGRAM

The Committee on Medical Motion Pictures has selected approximately twenty-five films on general surgery and the surgical specialties certain to be of great interest to Congress visitors. In addition to the regular surgical program special otolaryngology and ophthalmology films will be shown at their individual sessions Tuesday and Wednesday evenings.

HOTEL RESERVATIONS

Hotel accommodations are still available. For reservations send to Miss Sylvia Peltonen, Manager, American College of Surgeons Housing Bureau, 500 Park Avenue, New York 22, New York.

REGISTRATION

Official registration blanks, transportation information and hotel reservation forms have been sent to all Fellows. If reservations have not yet been made it is essential to do so immediately. Fees are as follows: Fellows whose dues are paid to December 1, 1952, no fee; endorsed candidates, $5.00; non-Fellows, $10.00; Initiates, no fee.

BERMUDA CRUISE

Reservations are still available for the nine day, post-Congress cruise to Bermuda on the luxurious *Queen of Bermuda*. Inquiries should be addressed to Mr. Leon V. Arnold, 36 Washington Square West, New York 11.

PROGRAM IN BRIEF

Monday, September 22, 1952

8:00–12:00 Daily Clinics
9:00–12:00 Medical Motion Pictures
10:00–11:00 General Assembly
11:00–12:30 Clinicopathology Conference
1:30– 3:00 Panel Discussion
1:30– 5:00 Daily Clinics
1:30– 5:30 Medical Motion Pictures (Davis & Geck)
1:30– 4:30 Television
2:00– 5:00 Forum on Fundamental Surgical Problems
2:00– 5:00 Forum—Surgical Specialty
3:30– 5:00 Panel Discussion
6:30– 7:00 Reception Preceding Presidential Dinner
7:00– 8:15 Presidential Dinner—Board of Regents, Wives, Guests
8:15– 8:30 Assembly for Officers, Regents, Guests
8:30–10:00 Presidential Meeting
8:00–10:00 Motion Picture Symposium

Tuesday, September 23, 1952

8:00–12:00 Daily Clinics and Postgraduate Courses
8:30–10:30 Breakfast, Committee on Nutrition
9:00–10:30 Panel Discussion, Ophthalmology
9:00–12:00 Television
9:00–12:00 Postgraduate Courses
9:00–12:00 Forum on Fundamental Surgical Problems (2 sections)
9:00–12:30 Ciné Clinic
10:45–12:15 Panel Discussion, Otolaryngology
1:00– 5:00 Medical Motion Pictures
1:30– 3:00 Panel Discussion
1:30– 4:30 Television
1:30– 5:00 Daily Clinics
2:00– 5:00 Symposium on Trauma
2:00– 5:00 Forum on Fundamental Surgical Problems
2:00– 5:00 Forum—Surgical Specialty
3:00– 5:00 Library Committee
3:30– 5:00 Panel Discussion
6:30– 8:00 Dinner for Speakers on Evening Programs
6:30–10:00 Dinner for Officers of Local Chapters
7:00– 8:00 Medical Motion Pictures (EENT)
8:30–10:30 Scientific Session, General Surgery
8:30–10:30 Scientific Session, Otolaryngology
8:30–10:30 Scientific Session, Ophthalmology
8:30–10:30 Motion Picture Symposium, Surgery of Heart

Wednesday, September 24, 1952

8:00–12:00 Daily Clinics
9:00–10:30 Panel Discussion, Ophthalmology
9:00–12:00 Television
9:00–12:00 Postgraduate Courses
9:00–12:00 Forum on Fundamental Surgical Problems (2 sections)
9:00–12:30 Ciné Clinic
10:00–12:00 Meetings of Credentials, Judiciary and Counseling Committees
10:45–12:15 Panel Discussion, Otolaryngology
1:00– 5:00 Medical Motion Pictures (Davis & Geck)
1:30– 3:00 Panel Discussion, General Surgery
1:30– 3:00 Panel Discussion, Gynecology and Obstetrics
1:30– 5:00 Daily Clinics
1:30– 5:30 Adjourned Meeting of Governors
1:30– 4:30 Television
2:00– 5:00 Symposium on Cancer
2:00– 5:00 Forum on Fundamental Surgical Problems
2:00– 5:00 Forum—Surgical Specialty
3:30– 5:00 Panel Discussion, General Surgery
3:30– 5:00 Panel Discussion, Gynecology and Obstetrics
3:30– 5:00 Meetings of Regional and National Trauma Committees
5:00– 6:00 International Society of Surgery
6:30– 8:00 Dinner for Speakers on Evening Programs
7:00– 8:00 Medical Motion Pictures (EENT)
8:30–10:30 Scientific Session
8:30–10:30 Scientific Session, Ophthalmology
8:30–10:30 Scientific Session, Otolaryngology

Thursday, September 25, 1952

7:30– 9:30 Breakfast Forum Committee
8:00–12:00 Daily Clinics and Postgraduate Courses
9:00–12:00 Television
9:00–10:30 Panel Discussion, Ophthalmology
9:00–12:00 Forum on Fundamental Surgical Problems (2 sections)
9:00–12:00 Postgraduate Courses
9:00–12:30 Ciné Clinic
10:00–12:00 Committee on Medical Motion Pictures
10:45–12:15 Panel Discussion, Otolaryngology
1:00– 5:30 Medical Motion Pictures
1:30– 3:00 Panel Discussion, General Surgery
1:30– 3:00 Panel Discussion, Gynecology and Obstetrics
1:30– 3:00 Forum on Fundamental Surgical Problems
1:30– 3:00 Forum—Surgical Specialty
1:30– 3:00 Daily Clinics
1:30– 3:00 Symposium, Graduate Training in Surgery
1:30– 4:30 Television
3:30– 3:45 Adjourned Meeting, Board of Governors
3:45– 6:00 Annual Meeting of Fellows
6:30– 8:00 Dinner for Speakers on Evening Programs
6:30–10:00 Annual Dinner Meeting, Committee on Trauma
7:00– 8:00 Medical Motion Pictures (EENT)
8:30–10:30 Scientific Session
8:30–10:30 Scientific Session, Ophthalmology
8:30–10:30 Scientific Session, Otolaryngology

Friday, September 26, 1952

8:00–12:00 Daily Clinics and Postgraduate Courses
8:30–10:30 p.m. Distribution and Checking of Fellowship Gowns
9:00–12:00 Television
9:00–12:00 Postgraduate Courses
9:00–12:00 Meeting, Board of Regents
9:00–12:00 Forum on Fundamental Surgical Problems (2 sections)
9:00–12:30 Ciné Clinic
12:00– 1:30 Luncheon, Board of Regents
1:30– 4:45 Panel Discussions:
Gynecology and Obstetrics
Neurologic Surgery
Orthopedic Surgery
Plastic Surgery
Thoracic Surgery
Urology
1:30– 3:00 Assembly of Initiates
6:30– 7:00 Reception Preceding Regents Dinner
6:30– 7:00 Reception Preceding Governors Dinner
7:00– 8:15 Regents Dinner—Board of Regents, Wives, Guests
7:00– 8:15 Governors Dinner
7:00– 8:15 Initiates Pre-Convocation Assembly
8:15– 8:30 Assembly for Officers, Regents, Governors, and Guests
8:30–10:00 Convocation

PRELIMINARY PROGRAM

GENERAL ASSEMBLY

Monday, 10:00—11:00 a.m., Grand Ballroom, Waldorf-Astoria

ALTON OCHSNER, M.D., F.A.C.S., New Orleans, President, American College of Surgeons, *Presiding.*
Greetings and Announcements. FRANK GLENN, M.D., F.A.C.S., New York; Chairman, New York Committee on Arrangements.
Inauguration of the Joint Hospital Accreditation Program
GUNNAR GUNDERSEN, M.D., F.A.C.S., Chairman, Joint Commission.
EDWIN L. CROSBY, M.D., Director.

CLINICOPATHOLOGY CONFERENCE

Monday, 11:00 a.m.—12:30 p.m., Grand Ballroom, Waldorf-Astoria

ALTON OCHSNER, M.D., F.A.C.S., New Orleans, President, American College of Surgeons, *Presiding.*
JOSEPH E. FLYNN, M.D., New York, Associate Professor of Pathology, Columbia University, *Moderator.*

PRESIDENTIAL MEETING

Monday, 8:30—10:00 p.m., Grand Ballroom, Waldorf-Astoria

ALTON OCHSNER, M.D., F.A.C.S., New Orleans; President, American College of Surgeons, *Presiding.*
Processional—Officers, Regents, and Guests.
Invocation.
Address of Welcome. FRANK GLENN, M.D., F.A.C.S., New York; Chairman, Committee on Arrangements.
Introduction of Distinguished Guests. EVARTS A. GRAHAM, M.D., F.A.C.S., St. Louis; Chairman, Board of Regents.
Address of the President. ALTON OCHSNER, M.D., F.A.C.S., New Orleans.
Inauguration of Officers:
Presented by THOMAS H. LANMAN, M.D., F.A.C.S., Boston; Retiring First Vice-President.
Second Vice-President: THOMAS F. MULLEN, M.D., F.A.C.S., San Francisco.
First Vice-President: ROBERT H. KENNEDY, M.D., F.A.C.S., New York.
President: HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pennsylvania.
The Seventh Martin Memorial Lecture: The Evolution of Gastric Surgery. DONALD C. BALFOUR, M.D., F.A.C.S., Rochester, Minnesota; Professor of Surgery, Mayo Foundation, Minnesota; Director Emeritus, Mayo Foundation for Medical Education and Research, Minnesota; Consulting Surgeon, Mayo Clinic.
Recessional.

CONVOCATION

Friday, 8:30—10:00 p.m., Grand Ballroom, Waldorf-Astoria

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pennsylvania; President, American College of Surgeons, *Presiding.*
Processional. Initiates, Governors, Officers, Regents, and Distinguished Guests.
Invocation. Most Reverend Joseph F. Flannelly, D.D., Auxiliary Bishop of New York.
Presentation of Initiates for Fellowship. Chairman of the Board of Regents.
Fellowship Pledge. The Initiates.
Conferring of Fellowships by the President. HAROLD L. FOSS, M.D., F.A.C.S.
Conferring of Honorary Fellowships. The President.
Fellowship Address: SIR CECIL WAKELEY, K.B.E.C.B., O.M., F.R.C.S. (England), F.R.A.C.S., President, Royal College of Surgeons, England.
Recessional.

EVENING SCIENTIFIC SESSIONS

GENERAL SURGERY

Tuesday, 8:30—10:30 p.m., Ballroom, Waldorf-Astoria

THOMAS F. MULLEN, M.D., F.A.C.S., San Francisco, *Presiding.*
Cytologic and Biopsy Diagnosis of Cancer. ARTHUR PURDY STOUT, M.D., New York.
Evaluation of Cancer Serodiagnostic Tests. ROBERT E. STOWELL, M.D., Kansas City, Kansas.
Cancer of the Lung. BRIAN B. BLADES, M.D., F.A.C.S., Washington, D.C.

Wednesday, 8:30—10:30 p.m., Ballroom, Waldorf-Astoria

ROBERT H. KENNEDY, M.D., F.A.C.S., New York, *Presiding.*
Trauma Oration. The Working Man's Hand. SUMNER L. KOCH, M.D., F.A.C.S., Chicago.
Medical and Surgical Management of Thromboembolic Disease
Medical Management of Thromboembolic Disease with an Evaluation of the Newer Anticoagulants. IRVING S. WRIGHT, M.D., F.A.C.P., New York.
The Management of Thromboembolic Disease in Surgical Patients. ARTHUR W. ALLEN, M.D., F.A.C.S., Boston.

Thursday, 8:30—10:30 p.m., Ballroom, Waldorf-Astoria

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pa., *Presiding.*
Laryngeal Sequelae of Intracheal Anesthesia. PAUL H. HOLINGER, M.D., F.A.C.S., Chicago.
Conservative Surgical Treatment of Endometriosis. JOE V. MEIGS, M.D., F.A.C.S., Boston.
Evaluation of Liver Function Tests. I. S. RAVDIN, M.D., F.A.C.S., Philadelphia.

OPHTHALMOLOGY

Tuesday, 9:00—10:30 a.m., Peacock Lounge, Waldorf-Astoria

Panel Discussion on "Problems Concerned with the Extraction of Complicated Cataracts"
Moderator: JOHN H. DUNNINGTON, M.D., F.A.C.S., New York.
Collaborators: WALTER S. ATKINSON, M.D., F.A.C.S., Watertown, New York; HENRY L. BIRGE, M.D., F.A.C.S., Hartford, Connecticut; GLEN GREGORY GIBSON, M.D., Philadelphia.

Tuesday, 8:30—10:30 p.m., Baroque Room, Belmont Plaza

EDWIN B. DUNPHY, M.D., F.A.C.S., Boston, *Presiding.*
Symposium on "The Surgical Correction of Motor Anomalies Involving the Extraocular Muscles"
Surgical Physiology of Extraocular Muscles. HAROLD W. BROWN, M.D., New York.
Surgical Correction of Esotropia. RICHARD G. SCOBEE, M.D., F.A.C.S., St. Louis.
Surgical Correction of Exotropia. HAROLD G. SCHEIE, M.D., F.A.C.S., Philadelphia.
Surgical Correction of Vertical Muscle Anomalies. WALTER H. FINK, M.D., F.A.C.S., Minneapolis.
Surgical Correction of Blepharoptosis. RAYNOLD N. BERKE, M.D., Hackensack, New Jersey.

Wednesday, 9:00—10:30 a.m., Peacock Lounge, Waldorf-Astoria

Panel Discussion on "Orbital Implants"
Moderator: ARNO E. TOWN, M.D., F.A.C.S., New York.
Collaborators: ALSTON CALLAHAN, M.D., F.A.C.S., Birmingham; JOHN P. MACNIE, M.D., F.A.C.S., New York; RAYMOND E. MEEK, M.D., F.A.C.S., New York.

Wednesday, 8:30—10:30 p.m., Moderne Room, Belmont Plaza

WENDELL L. HUGHES, M.D., F.A.C.S., Hempstead, New York, *Presiding.*
Symposium on "Causes of Failure in Ocular Surgical Procedures"
Pathological Causes of Failure Following Intraocular Operations. BRITTAIN FORD PAYNE, M.D., F.A.C.S., New York.
Failure in Glaucoma Operations. JOSEPH A. C. WADSWORTH, M.D., New York.
Failure in Cataract Operations. DANIEL B. KIRBY, M.D., F.A.C.S., New York.
Failure in Extraocular Muscle Surgery. TRUMAN L. BOYES, M.D., F.A.C.S., New York.
Failure in Retinal Detachment Surgery. P. ROBB MCDONALD, M.D., F.A.C.S., Philadelphia.

Thursday, 9:00—10:30 a.m., Peacock Lounge, Waldorf-Astoria

Panel Discussion on "Fallacies in Refraction by Use of Mechanical Refractors"
Moderator: CONRAD BERENS, M.D., F.A.C.S., New York.

Collaborators: ALFRED COWAN, M.D., Philadelphia; ARTHUR LINKSZ, M.D., F.A.C.S., New York; DANIEL SNYDACKER, M.D., Chicago.

Thursday, 8:30—10:30 p.m., Moderne Room, Belmont Plaza

FREDERICK H. VERHOEFF, M.D., F.A.C.S., Boston, *Presiding.*
Sympathetic Ophthalmia. HAROLD H. JOY, M.D., F.A.C.S., Syracuse, New York.
Discusser: GEORGE M. HAIK, M.D., F.A.C.S., New Orleans.
Atomic Eye Injuries. ELBERT DE COURSEY, Brigadier General, M.C., U.S.A., Washington, D.C.
Discusser: DAVID G. COGAN, M.D., Boston.
Management of Intraocular Foreign Bodies. RAYMOND L. PFEIFFER, M.D., F.A.C.S., New York.
Discusser: NORMAN L. CUTLER, M.D., Wilmington, Delaware.
Treatment of Eye Burns. ROBISON D. HARLEY, M.D., F.A.C.S., Atlantic City, New Jersey.
Discusser: HEDWIG S. KUHN, M.D., Hammond, Indiana.

OTOLARYNGOLOGY

Tuesday, 10:45 a.m.—12:15 p.m., Peacock Lounge, Waldorf-Astoria

LEROY A. SCHALL, M.D., F.A.C.S., Boston, *Presiding.*
Teratological Lesions of the Head and Neck. ALBERT C. FURSTENBERG, M.D., F.A.C.S., Ann Arbor.
Discusser: JOHN J. CONLEY, M.D., F.A.C.S., New York.
Panel Discussion on "Nasopharyngeal Fibromata"
Moderator: LEROY A. SCHALL, M.D., F.A.C.S., Boston.
Collaborators: FREDERICK A. FIGI, M.D., F.A.C.S., Rochester, Minnesota; PERCY E. IRELAND, M.D., Toronto, Ontario; JAMES H. MAXWELL, M.D., F.A.C.S., Ann Arbor, Michigan.

Tuesday, 8:30—10:30 p.m., Baroque Room, Belmont Plaza

FREDERICK A. FIGI, M.D., F.A.C.S., Rochester, Minnesota, *Presiding.*
Plasma Substitutes. LAMAR SOUTTER, M.D., F.A.C.S., Boston.
Mycotic Infections. E. KING GILL, M.D., F.A.C.S., Corpus Christi, Texas.
Discusser: DOUGLAS P. TORRE, M.D., New York.
Treatment of Hemangiomas. FRED Z. HAVENS, M.D., F.A.C.S., Rochester, Minnesota.

Wednesday, 10:45 a.m.—12:15 p.m., Peacock Lounge, Waldorf-Astoria

JAMES H. MAXWELL, M.D., F.A.C.S., Ann Arbor, *Presiding.*
Modified Radical Mastoidectomy. ARTHUR L. JUERS, M.D., Miami.
Discusser: J. BROWN FARRIOR, M.D., F.A.C.S., Tampa.
Infectious Mononucleosis. CHRIS J. S. ZARAFONETIS, M.D., Philadelphia.
Discusser: JOHN E. BORDLEY, M.D., Baltimore.

Wednesday, 8:30—10:30 p.m., Baroque Room, Belmont Plaza

PAUL H. HOLINGER, M.D., F.A.C.S., Chicago, *Presiding.*
Symposium on "Re-evaluation of Antibiotics"
General Uses in Surgery. CHAMP LYONS, M.D., F.A.C.S., Birmingham.
Uses and Abuses of Antibiotics in Relation to Allergic and Other Reactions. PERRIN LONG, M.D., F.R.C.P., Brooklyn.
Specific Problems in the Use of Antibiotics in Otolaryngology. HARRY P. SCHENCK, M.D., F.A.C.S., Philadelphia.
Sensitivity Tests. BALBINA A. JOHNSON, New York.

Thursday, 10:45 a.m.—12:15 p.m., Peacock Lounge, Waldorf-Astoria

HARRY P. SCHENCK, M.D., F.A.C.S., Philadelphia, *Presiding.*
Respiratory Obstructions in Infants, Including Atelectasis in the Newborn. PAUL H. HOLINGER, M.D., F.A.C.S., Chicago.
Differential Diagnosis of Wheezing Respirations. LOUIS H. CLERF, M.D., F.A.C.S., Philadelphia.
Management of Respiratory Difficulty in Bulbar-Poliomyelitis. THOMAS C. GALLOWAY, M.D., Evanston, Illinois.

Thursday, 8:30—10:30 p.m., Baroque Room, Belmont Plaza

LOUIS H. CLERF, M.D., F.A.C.S., Philadelphia, *Presiding.*
Symposium on "Operating Room Emergencies"
Respiratory Failure (Including Pathological Physiology of Anesthesia). CARL A. MOYER, M.D., F.A.C.S., St. Louis.

Acute Adrenal Cortical Failure. LOUIS J. SOFFER, M.D., New York.
Cardiac Arrest. FRANK B. BERRY, M.D., F.A.C.S., New York.

PANEL DISCUSSIONS

GENERAL SURGERY

Monday, 1:30—3:00 p.m., Grand Ballroom, Waldorf-Astoria

Choice of Operation for Cancer of the Rectum
Moderator: FRED W. RANKIN, M.D., F.A.C.S., Lexington.
Collaborators: CLAUDE F. DIXON, M.D., F.A.C.S., Rochester, Minnesota; R. KENNEDY GILCHRIST, M.D., F.A.C.S., Chicago; JESSIE C. GRAY, M.D., F.A.C.S., Toronto.

3:30—5:00 p.m.

Blood, Plasma and Plasma Expanders
Moderator: CHAMP LYONS, M.D., F.A.C.S., Birmingham.
Collaborators: ROBERT ELMAN, M.D., F.A.C.S., St. Louis; DAVID METHENY, M.D., F.A.C.S., Seattle; JONATHAN E. RHOADS, M.D., F.A.C.S., Philadelphia.

Tuesday, 1:30—3:00 p.m., Grand Ballroom, Waldorf-Astoria

Gallbladder Disease
Moderator: NATHAN W. WOMACK, M.D., F.A.C.S., Chapel Hill, North Carolina.
Collaborators: D. L. C. BINGHAM, M.D., F.A.C.S., Kingston, Ontario; GEORGE CRILE, JR., M.D., F.A.C.S., Cleveland; ANGUS D. MCLACHLIN, M.D., F.A.C.S., London, Ontario.

3:30—5:00 p.m.

Carcinoma of the Stomach
Moderator: LELAND S. MCKITTRICK, M.D., F.A.C.S., Brookline, Massachusetts.
Collaborators: BENEDICTO MONTENEGRO, M.D., F.A.C.S., Sao Paulo, Brazil; H. ROCKE ROBERTSON, M.D., F.A.C.S., Vancouver, B.C.; ROBERT M. ZOLLINGER, M.D., F.A.C.S., Columbus, Ohio.

Wednesday, 1:30—3:00 p.m., Grand Ballroom, Waldorf-Astoria

Pancreatic Disease
Moderator: I. MIMS GAGE, M.D., F.A.C.S., New Orleans.
Collaborators: KENNETH E. LEMMER, M.D., F.A.C.S., Madison, Wisconsin; WILLIAM F. RIENHOFF, JR., M.D., F.A.C.S., Baltimore; HARRIS B. SHUMACKER, JR., M.D., F.A.C.S., Indianapolis.

3:30—5:00 p.m.

Postphlebitic Syndrome
Moderator: ROBERT R. LINTON, M.D., F.A.C.S., Brookline, Massachusetts.
Collaborators: ROBERT W. BUXTON, M.D., F.A.C.S., Ann Arbor; PAUL T. DECAMP, M.D., F.A.C.S., New Orleans; J. C. LUKE, M.D., F.A.C.S., Montreal.

Thursday, 1:30—3:00 p.m., Grand Ballroom, Waldorf-Astoria

Current Practice in Managing Water, Electrolyte, and Protein Balance Problems in Surgical Patients
Moderator: JOHN H. MULHOLLAND, M.D., F.A.C.S., New York.
Collaborators: ERNEST S. BREED, M.D., New York; CHARLES G. CHILD III, M.D., F.A.C.S., New York; DAVID V. HABIF, M.D., F.A.C.S., New York; HENRY T. RANDALL, M.D., New York.

GYNECOLOGY AND OBSTETRICS

Wednesday, 1:30—3:00 p.m., Ballroom, Biltmore

Pain in the Right Lower Quadrant of the Female Abdomen
Moderator: JOHN C. BURCH, M.D., F.A.C.S., Nashville.
Collaborators: R. GORDON DOUGLAS, M.D., New York; CHARLES C. HIGGINS, M.D., F.A.C.S., Cleveland; JONATHAN E. RHOADS, M.D., F.A.C.S., Philadelphia.

3:30—5:00 p.m.

Urologic Survey Before and After Gynecologic Surgery
Moderator: J. MASON HUNDLEY, JR., M.D., F.A.C.S., Baltimore.

Collaborators: WILLIAM K. DIEHL, M.D., F.A.C.S., Baltimore; HOUSTON S. EVERETT, M.D., Baltimore; WALTER S. KERR, JR., M.D., Boston.

Thursday, 1:30—3:00 p.m., Ballroom, Biltmore

Management of the Rh Negative Patient
Moderator: NEWELL W. PHILPOTT, M.D., F.A.C.S., Montreal.
Collaborators: CARL BACHMAN, M.D., F.A.C.S., Philadelphia; RONALD L. DENTON, M.D., F.R.C.P., Montreal; PETER VOGEL, M.D., New York.

Friday, 1:30—3:00 p.m., Ballroom, Biltmore

The Present Status of the Vaginal Smear in Diagnosis of Malignancy
Moderator: LEWIS C. SCHEFFEY, M.D., F.A.C.S., Philadelphia.
Collaborators: EMERSON DAY, M.D., New York; ARTHUR T. HERTIG, M.D., Boston; GEORGE N. PAPANICOLAOU, M.D., New York; A. E. RAKOFF, M.D., Philadelphia.

3:15—4:45 p.m.

Diagnostic Procedure and Treatment of Nonmalignant Lesions Causing Bleeding
Moderator: GEORGE V. SMITH, M.D., F.A.C.S., Brookline, Massachusetts.
Collaborators: GEORGE E. JUDD, M.D., F.A.C.S., Los Angeles; EMIL NOVAK, M.D., F.A.C.S., Baltimore; HOWARD ULFELDER, M.D., F.A.C.S., Boston.

SURGICAL SPECIALTIES

NEUROLOGIC SURGERY

Friday, 1:30—3:00 p.m., Music Room, Biltmore

The Treatment of Intracranial Glioblastomas
Moderator: HARRY WILKINS, M.D., F.A.C.S., Oklahoma City.
Collaborators: THEODORE C. ERICKSON, M.D., Madison, Wisconsin; LEONARD T. FURLOW, M.D., St. Louis; STANTON L. GOLDSTEIN, M.D., Hines, Illinois.

3:15—4:45 p.m.

The Diagnosis and Results of Treatment of Herniated Nucleus Pulposus in the Cervical Spinal Canal
Moderator: HENRY G. SCHWARTZ, M.D., F.A.C.S., St. Louis.
Collaborators: EDGAR F. FINCHER, M.D., F.A.C.S., Emory University, Georgia; JAMES GREENWOOD, JR., M.D., F.A.C.S., Houston, Texas; WILLIAM B. SCOVILLE, M.D., F.A.C.S., Hartford, Connecticut.

ORTHOPEDIC SURGERY

Friday, 1:30—3:00 p.m., Empire Room, Waldorf-Astoria

Indications and Technique for Internal Fixation, Including Use of the Intramedullary Nail, of Fractures of the Shaft of the Femur
Moderator: HUGH SMITH, M.D., F.A.C.S., Memphis.
Collaborators: JAMES J. CALLAHAN, M.D., F.A.C.S., Chicago; JULIAN E. JACOBS, M.D., F.A.C.S., Charlotte, North Carolina; JAMES E. M. THOMSON, M.D., F.A.C.S., Lincoln, Nebraska.

3:15—4:45 p.m.

Indications and Technique for Internal Fixation, Including Use of the Intramedullary Nail, of Fractures of the Tibia
Moderator: FRED C. REYNOLDS, M.D., F.A.C.S., St. Louis.
Collaborators: EDWIN F. CAVE, M.D., F.A.C.S., Boston; EDWARD C. HOLSCHER, M.D., F.A.C.S., St. Louis; JAMES K. STACK, M.D., F.A.C.S., Chicago; J. OTTO LOTTES, M.D., St. Louis.

PLASTIC SURGERY

Friday, 1:30—3:00 p.m., Baroque Room, Belmont Plaza

The Treatment of Open Injuries Complicated by Extensive Loss of Covering Tissue
Moderator: SUMNER L. KOCH, M.D., F.A.C.S., Chicago.
Collaborators: WILLIAM H. FRACKELTON, M.D., F.A.C.S., Milwaukee; STUART DOUGLAS GORDON, M.D., F.A.C.S., Toronto; J. WILLIAM LITTLER, M.D., New York; FRANK MCDOWELL, M.D., F.A.C.S., St. Louis; GEORGE V. WEBSTER, M.D., F.A.C.S., Pasadena.

3:15—4:45 p.m.

The Replacement of Extensive Scars of the Lower Extremity with Sliding, Cross Leg, and Distant Pedicle Flaps
Moderator: S. MILTON DUPERTUIS, M.D., F.A.C.S., Pittsburgh.
Collaborators: ALFRED W. FARMER, M.D., Toronto; W. BRANDON MACOMBER, M.D., F.A.C.S., Albany; RADFORD C. TANZER, M.D., Hanover, N.H.

THORACIC SURGERY

Friday, 1:30—3:00 p.m., Sert Room, Waldorf-Astoria

The Problem of Cardiac Arrest; Its Prevention and Treatment
Moderator: JOHN H. GIBBON, JR., M.D., F.A.C.S., Philadelphia.
Collaborators: W. ANDREW DALE, M.D., Rochester, N.Y.; FRANK GLENN, M.D., F.A.C.S., New York; EMANUEL M. PAPPER, M.D., New York.

3:15—4:45 p.m.

Recent Developments in the Surgical Treatment of Pulmonary Tuberculosis
Moderator: JOHN ALEXANDER, M.D., F.A.C.S., Ann Arbor, Michigan.
Collaborators: J. BURNS AMBERSON, M.D., New York; JOSEPH W. GALE, M.D., F.A.C.S., Madison, Wisconsin; ROBERT R. SHAW, M.D., Dallas, Texas.

UROLOGY

Friday, 1:30—3:00 p.m., Bowman Room, Biltmore

Management of Surgical Injuries to the Urinary Tract
Moderator: VICTOR F. MARSHALL, M.D., F.A.C.S., New York.
Collaborators: AUSTIN I. DODSON, M.D., F.A.C.S., Richmond; WILLIAM J. ENGEL, M.D., F.A.C.S., Cleveland; LAWRENCE R. WHARTON, M.D., Baltimore; WILLET F. WHITMORE, JR., M.D., F.A.C.S., New York.

3:15—4:45 p.m.

Management of the Congenital Anomalies of the Urinary Tract
Moderator: VICTOR F. MARSHALL, M.D., F.A.C.S., New York.
Collaborators: EDGAR BURNS, M.D., F.A.C.S., New Orleans; NORRIS J. HECKEL, M.D., F.A.C.S., Chicago; CHARLES C. HIGGINS, M.D., F.A.C.S., Cleveland; GERSHOM J. THOMPSON, M.D., F.A.C.S., Rochester, Minnesota.

SYMPOSIUM ON TRAUMA

Tuesday, 2:00—5:00 p.m., Ballroom, The Biltmore

R. ARNOLD GRISWOLD, M.D., F.A.C.S., Louisville, Chairman, Committee on Trauma, American College of Surgeons, *Presiding.*

Acute Intervertebral Disc Lesions in the Lower Cervical Region. R. GLEN SPURLING, M.D., F.A.C.S., Louisville, and EVERETT G. GRANTHAM, M.D., F.A.C.S., Louisville.

Nonunion of Fractures. ROBERT P. KELLY, JR., M.D., F.A.C.S., Emory University.

Intra-arterial Transfusion in Trauma. SAM F. SEELEY, Brigadier General (M.C.) U.S.A., F.A.C.S., Washington.

Anesthesia in Trauma. HENRY K. BEECHER, M.D., Boston.

Intramedullary Nailing of Fractures. DENMAN C. HUCHERSON, M.D., F.A.C.S., Houston.

Evaluation of Exposure Method of Local Treatment in Major Second and Third Degree Burns. (A Preliminary Report Analyzing This Treatment of the Burn Wound in Forty-Five Patients.) LOUIS M. ROUSSELOT, M.D., New York; JAMES F. CONNELL, JR., M.D., New York; WILLIAM P. WHALEN, M.D., New York.

MOTION PICTURE SYMPOSIUM ON SURGERY OF THE HEART

Tuesday, 8:00—10:00 p.m., Empire Room, Waldorf-Astoria

SYMPOSIUM ON CANCER

Wednesday, 2:00—5:00 p.m.

EDWIN P. LEHMAN, M.D., F.A.C.S., Charlottesville, Chairman, Committee on Cancer, American College of Surgeons, *Presiding.*

Treatment of Rectal Polypi Showing Doubtful or Small Malignant Changes. RUPERT B. TURNBULL, JR., M.D., Cleveland.

Cancer Detection; a Realistic Appraisal of its Value. IAN MACDONALD, M.D., F.A.C.S., Los Angeles.

The Combined Procedure for Excision of Intraoral and Hypopharyngeal Cancer with Radical Dissection. HAYES E. MARTIN, M.D., F.A.C.S., New York.

Carcinoma of the Pancreas. RICHARD B. CATTELL, M.D., F.A.C.S., Boston.

Internal Mammary Node Dissection Combined with Radical Mastectomy for Cancer of the Breast. F. JOHN LEWIS, M.D., University of Minnesota Hospitals, Minneapolis. OWEN H. WANGENSTEEN, M.D., F.A.C.S., Minneapolis.

GRADUATE TRAINING SYMPOSIUM

Thursday, 1:30—3 p.m., Bowman Room, The Biltmore

FREDERICK A. COLLER, M.D., F.A.C.S., Ann Arbor, Chairman, Graduate Training Committee, *Moderator.*

Symposium on "Residency Problems in Smaller Hospitals"

General Surgery. LAWRENCE CHAFFIN, M.D., F.A.C.S., Los Angeles.

Urology. GILBERT J. THOMAS, M.D., F.A.C.S., Santa Monica.

Orthopedic Surgery. GUY CALDWELL, M.D., F.A.C.S., New Orleans.

Otolaryngology. HARRY P. SCHENCK, M.D., F.A.C.S., Philadelphia.

Obstetrics and Gynecology. SAMUEL A. COSGROVE, M.D., F.A.C.S., Jersey City.

ANNUAL MEETING—BOARD OF GOVERNORS

Luncheon, Sunday, 12:30—5:00 p.m., LePerroquet Suite, Waldorf-Astoria

WILLIAM L. ESTES, JR., M.D., F.A.C.S., Bethlehem, Pennsylvania, Chairman, *Presiding.*

Minutes of 1951 Annual Meeting of the Board of Governors. GRANTLEY W. TAYLOR, M.D., F.A.C.S., Boston, Secretary.

Report of the Executive Committee. WILLIAM L. ESTES, JR., M.D., F.A.C.S., Chairman.

Report of the President. ALTON OCHSNER, M.D., F.A.C.S., New Orleans.

Report of the Chairman of the Board of Regents. EVARTS A. GRAHAM, M.D., F.A.C.S., St. Louis.

Report of The Director. PAUL R. HAWLEY, M.D., Chicago.

Hospital Accreditation.

Inter-American Relations of the College.

Group Insurance.

Report on Graduate Training Program. FREDERICK A. COLLER, M.D., F.A.C.S., Ann Arbor, Chairman, and GEORGE W. STEPHENSON, M.D., F.A.C.S., Assistant Director, Chicago.

Report on Sectional Meetings. H. PRATHER SAUNDERS, M.D., F.A.C.S., Associate Director, Chicago.

Report of Assistant Director. GEORGE W. STEPHENSON, M.D., F.A.C.S., Chicago.

Fellowship in the College.

Blue Shield Insurance Problems.

Reports from Governors Who Were Nominated by Special Societies or Federal Services.

Resolutions from Members of Board of Governors.

Reports from Governors Concerning Local Chapters.

Announcement of Meeting—Credentials, Judiciary, and Counseling Committees, 10:00 a.m. September 24.

FIRST ADJOURNED ANNUAL MEETING—BOARD OF GOVERNORS

Wednesday, 1:30—5:30 p.m., LePerroquet Suite, Waldorf-Astoria

Unfinished Business.

New Business.

Adjournment.

SECOND ADJOURNED ANNUAL MEETING—BOARD OF GOVERNORS

Thursday, 3:30—3:45 p.m., Grand Ballroom, Waldorf-Astoria

Report of the Committee on Nomination:
Election of Officers of the Governors.
Election of Regents of the College.
Appointment of Advisory Committee on Nominations of the Board of Governors for 1952–1953.
Adjournment.

ANNUAL MEETING OF FELLOWS

Thursday, 3:45—6:00, Grand Ballroom, Waldorf-Astoria

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pa.; President, *Presiding.*
Report of the Committee on Nominations:
Election of Officers of the College.
Election of Governors of the College.
Deliberations of the Board of Governors—1952 Annual Meeting. WILLIAM L. ESTES, JR., M.D., F.A.C.S., Bethlehem, Pennsylvania, Chairman, Board of Governors.
Report of the Treasurer. WARREN H. COLE, M.D., F.A.C.S., Chicago; Treasurer.
Cancer Committee. EDWIN P. LEHMAN, M.D., F.A.C.S., Charlottesville, Va.; Chairman.
Committee on Trauma. R. ARNOLD GRISWOLD, M.D., F.A.C.S., Louisville; Chairman.
Committee on Graduate Training in Surgery. FREDERICK A. COLLER, M.D., F.A.C.S., Ann Arbor; Chairman.
Committee on Medical Motion Pictures. HILGER P. JENKINS, M.D., F.A.C.S., Chicago; Chairman.
Committee on Nutrition of the Surgical Patient. WALTER G. MADDOCK, M.D., F.A.C.S., Chicago; Chairman.
The American College of Surgeons—Summary of Annual Reports of Departments and Committees. PAUL R. HAWLEY, M.D., Chicago; The Director, American College of Surgeons.
General Discussion.
Adjournment.

FUTURE CLINICAL CONGRESSES

1953	Chicago, Illinois	October 5 to 9
1954	Atlantic City, New Jersey	October 25 to 29
1955	Chicago, Illinois	October 31 to November 4

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

The Stomach

Monday, 9:00 a.m.–12:00, Sert Room, Waldorf-Astoria

Comparison of Insulin and Gastrometric Tests for Completeness of Vagotomy. C. R. ROWE, JR., KEITH S. GRIMSON, M.D., F.A.C.S., and B. H. FLOWE, Durham, North Carolina. Department of Surgery, Duke University School of Medicine.

Quantitative Studies on Effect of Gastrojejunostomy on Gastric Secretion. JOSÉ MA. ZUBIRAN, M.D., ALLAN E. KARK, M.D., ANTONIO J. MONTALBETTI, M.D., and LESTER R. DRAGSTEDT, M.D., Ph.D., F.A.C.S., Chicago, Illinois. Department of Surgery, University of Chicago, The School of Medicine of the Division of Biological Sciences.

Secretory Response of Stomach to Gastroenterostomy as Measured by a Heidenhain Pouch; an Experimental Study. EDMUND A. KANAR, M.D., EVERETT J. SCHMITZ, M.D., LESTER R. SAUVAGE, M.D., EDWARD H. STORER, and HENRY N. HARKINS, M.D., F.A.C.S., Seattle, Washington. Department of Surgery, University of Washington College of Medicine.

Effect of Vagotomy of the Main Stomach on Heidenhain Pouch Secretion. EVERETT J. SCHMITZ, M.D., EDMUND A. KANAR, M.D., LESTER R. SAUVAGE, M.D., EDWARD H. STORER, and HENRY N. HARKINS, M.D., F.A.C.S., Seattle, Washington. Department of Surgery, University of Washington School of Medicine.

Investigation of Irradiation Changes in Gastric Mucosa Utilizing Intragastric Balloons and Direct Exposure of Transplanted Mucosa. GEORGE E. MOORE, M.D., CLAUDE R. HITCHCOCK, M.D., and GRAFTON A. SMITH, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School.

Evaluation of Mucolytic Agents in Gastric Cytologic Studies. HERBERT F. TRAUT, M.D., F.A.C.S., MILTON ROSENTHAL, M.D., JAMES T. HARRISON, SEYMOUR M. FARBER, M.D., and ORVILLE F. GRIMES, M.D., San Francisco, California. University of California Medical Center.

Gastric Secretory Responses to Surgical Stress and Infection. CHESTER W. HOWE, M.D., F.A.C.S., WILLIAM C. WIGGLESWORTH, M.D., and WILLIAM J. PORELL, M.D., Boston, Massachusetts. Surgical Service, Smithwick Foundation, Massachusetts Memorial Hospitals, and Department of Surgery, Boston University School of Medicine.

An Experimental Study of the Nutrition of Animals Following Gastrectomy. ALLEN JOHNSON, M.D., PHILIP GOLDMAN, M.D., COOPER DAVIS, M.D., HAROLD HARPER, Ph.D., and HORACE J. MCCORKLE, M.D., F.A.C.S., San Francisco, California. Experimental Laboratories, University of California School of Medicine.

An Experimental Evaluation of the Billroth I Operation with Vagotomy. THOMAS W. MORGAN, M.D., Columbus, Ohio. Department of Surgery and Surgical Laboratories, Ohio State University College of Medicine.

Metabolic Intake-Excretion Study in Post Subtotal Gastrectomy Invalidism. PAUL C. KIERNAN, M.D., F.A.C.S., JUANITA HAYMOND, M.A., A.D.A., and STEPHEN KOZIOL, B.S. Washington, D.C., Georgetown University and Emergency Hospitals.

Effect of Partial Excision of the Pyloric Antrum on Acid Secretion in Heidenhain Pouch Dogs. RAYMOND HELSBY, M.B., F.R.C.S. (Eng.), and DONALD S. AUTEN, M.D., Chicago, Illinois. Department of Surgery, University of Illinois College of Medicine.

Evaluation of Tubular Gastric Resection Combined with Transverse Gastroplasty as Suitable Operation for Peptic Ulcer. WILLIAM D. KELLY, M.D., SNORRI HALL GRIMSON, M.D., and RICHARD EGDAHL, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School.

Esophagus and Intestine

Monday, 2:00–5:00 p.m., Sert Room, Waldorf-Astoria

Further Experimental Evaluation in the Dog of Esophagogastrectomy for the High-Lying Gastric Ulcer. LAWRENCE B. KIRILUK, M.D., and K. ALVIN MERENDINO, M.D., F.A.C.S., Seattle, Washington. Department of Surgery, University of Washington School of Medicine.

Use of Colon to Replace Lower Esophagus in Man: Report of a Case Four Years After Operation. CHARLES W. ROBERTSON, M.D., F.A.C.S., CHESTER W. HOWE, M.D., F.A.C.S., REGINALD H. SMITHWICK, M.D., F.A.C.S., Boston, Massachusetts. Departments of Surgery, Massachusetts Memorial Hospitals, Boston University School of Medicine.

Extent to Which One May Interfere with Blood Supply of Esophagus of the Dog. Follow-up Study. JOSEPH E. MACMANUS, M.D., F.A.C.S., JOSEPH T. DAMERON, M.D., JOHN R. PAINE, M.D., F.A.C.S., DAVID D. DUGAN, M.D., F.A.C.S., and RICHARD W. EGAN, M.D., Buffalo, New York. Buffalo General Hospital.

Repair of Experimental Lesions of Esophagus with Skin Transplants. L. H. RUBENSTEIN, M.D., and N. C. JEFFERSON, M.D., Chicago, Illinois. Department of Gastrointestinal Research, Medical Research Institute of Michael Reese Hospital, and Department of Surgery, Chicago Medical School.

Bridging of Esophageal Defects with Fresh and Preserved Aorta Grafts. HUSHANG JAVID, M.D., Chicago. Department of Surgery, University of Illinois College of Medicine.

Determination of Intestinal Viability; an Experimental Evaluation of Multiple Tests. IRVING ENQUIST, M.D., and ARNOLD J. KREMEN, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School, and University Surgical Service of Mt. Sinai Hospital.

Relationship of Lysozyme to the Mechanism and Cause of Death in Intestinal Obstruction. EARL EDWARD WILKISON, M.D., Charlottesville, Virginia. Department of Surgery, University of Virginia Department of Medicine.

Further Studies on Abnormal Hemin Pigment Found in Strangulation Obstruction. PAUL NEMIR, JR., M.D., HERBERT R. HAWTHORNE, M.D., F.A.C.S., and DAVID L. DRABKIN, M.D., Philadelphia, Pennsylvania. The Harrison Department of Surgical Research and Department of Physiological Chemistry, Graduate School of Medicine, University of Pennsylvania.

Strangulation Obstruction; the Effect of Diverting Duodenal Contents. ISIDORE COHN, JR., M.D., ALBERT GELB, M.D., HERBERT R. HAWTHORNE, M.D., F.A.C.S., and DAVID L. DRABKIN, M.D., Philadelphia, Pennsylvania. The Harrison Department of Surgical Research, Department of Surgery, Graduate School of Medicine, University of Pennsylvania.

Effect of Increased Atmospheric Pressures on Viability of Bowel Wall and Absorption of Gas in Closed Loop Obstructions. FREDERICK S. CROSS, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School.

Primary Resection of Colon in Fulminating Ulcerative Colitis. CHARLES B. RIPSTEIN, M.D., F.R.C.S. (Can.), F.A.C.S., New York. Department of Surgery, Maimonides Hospital of Brooklyn, State University of New York College of Medicine.

Clinical and Experimental Work on Massive Resection of Small Intestine. B. N. MARIEN, M.D., and JAMES R. MCCORRISTON, M.D., M.Sc., Montreal, Canada. Department of Experimental Surgery, McGill University Faculty of Medicine.

The Heart and Great Vessels

Tuesday, 9:00 a.m.–12:00, Sert Room, Waldorf-Astoria

Long-Term Studies of Fate and Function of Maximal-Length Vena Cava Autografts to Bridge Experimental Aortic Defects in Dogs. ROBERT A. NABATOFF, M.D., ARTHUR S. W. TOUROFF, M.D., F.A.C.S., AND MILTON GROSS, B.A., New York. Surgical Service of Mt. Sinai Hospital.

Long-Term Observations on Autogenous Pericardial and Venous Grafts in Thoracic Aorta. YOSHIO SAKO, M.D., and RICHARD L. VARCO, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School.

Two-Year Postoperative Study of Thoracic Arterial Grafts Transplanted Into Puppies. EDWARD B.C. KEEFER, M.D., FRANK GLENN, M.D., F.A.C.S., and CHARLES T. DOTTER, M.D., New York, New York. Departments of Surgery and Radiology of the New York Hospital, Cornell Medical Center.

Early Results in Experimental Use of Freeze-Dried Arterial Grafts. JAMES W. PATE (Lt. j.g., M.C., USNR), PHILIP N. SAWYER (Lt. j.g., M.C., USNR), Bethesda, Maryland, RALPH A. DETERLING, JR., M.D., F.A.C.S., J. WALLACE BLUNT, M.D., and MARY S. PARSHLEY, Ph.D., New York, New York. Naval Medical Research Institute, and Tissue Bank, Naval Medical School, National Naval Medical Center, Bethesda, Maryland, and the Department of Surgery, Columbia University College of Physicians and Surgeons, New York.

Direct Anastomosis Versus Ligation in Traumatic Arteriovenous Fistulas and Aneurysms. Experience with 132 Consecutive Korean Wounded. SAM F. SEELEY, M.D., F.A.C.S. (Brig. Gen., M.C., USA), CARL W. HUGHES M.D., (Lt. Col., M.C., USA), EDWARD J. JAHNKE, M.D. (Capt., M.C., USAF), Washington, D.C. Surgical Service, Walter Reed Army Medical Center.

An Oxygenator For Use in a Heart-Lung Apparatus. NICHOLAS S. GIMBEL, M.D., and JOSEPH ENGELBERG, Philadelphia, Pennsylvania. The Harrison Department of Surgical Research, University of Pennsylvania School of Medicine.

Discussion of Total By-pass of Heart and Lungs in Dogs by Means of Pump-Oxygenator. JAMES A. HELMSWORTH, M.D., F.A.C.S., ROGER T. SHERMAN, M.D., SAMUEL KAPLAN, M.D., LELAND C. CLARK, JR., Ph.D., and THOMAS LARGEN, M.D., Cincinnati, Ohio. Department of Surgery, University of Cincinnati College of Medicine, and Department of Pediatrics, The Fels Institute for Research in Human Development, Antioch College, Yellow Springs, Ohio.

Acute Metabolic Changes Associated with Employment of Pump-Oxygenator to Supplant Heart and Lungs. DWIGHT S. SPRENG, JR., M.D., CLARENCE DENNIS, M.D., Ph.D., F.A.C.S., LAVONNE YOUNG, B.S., and GEORGE E. NELSON, M.D., New York. University of Minnesota Medical School, and State University of New York Medical Center at New York City.

Further Studies on Survival of Transplanted Heart. EMANUEL MARCUS, M.D., Ph.D., ALDO LUISADA, M.D., SAMUEL N. T. WONG, M.D., and W. C. LIU, M.D., Chicago, Illinois. Chicago Medical School.

Tolerance of Heart to Temporary Complete Vena Caval Occlusion. ROBERT N. HAMMERSTROM, M.D., MITCHELL SPELLMAN, M.D., MORLEY COHEN, M.D., RICHARD L. VARCO, M.D., and C. WALTON LILLEHEI, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School.

Fate of Intracardiac Pericardial Grafts as Applied to Closure of Septal Defects and to Relief of Mitral Insufficiency. ROBERT P. GLOVER, M.D., ALFRED R. HENDERSON, M.D., RUY MARGUTTI, M.D., and JOHN GREGORY, M.D., Philadelphia, Pennsylvania.

Pressure Gradient Across the Interatrial Septum Throughout Cardiac Cycle; an Experimental Study in Normal Dogs. E. W. HOAG, M.D., R. A. BRUCE, M.D., C. A. WIEDERHIELM, and K. ALVIN MERENDINO, M.D., F.A.C.S., Seattle, Washington. Department of Surgery, University of Washington School of Medicine.

Study of Hemodynamics and Closure of Experimental Interventricular Septal Defects. GEORGE H. A. CLOWES, JR., M.D., ROBERT H. WHITTLESEY, M.D., and WILLIAM E. NEVILLE, M.D., F.A.C.S., Cleveland, Ohio. Western Reserve University School of Medicine and Department of Surgery, Cleveland City Hospital.

Wounds and Infection

Tuesday, 9 a.m.–12 noon, Empire Room, Waldorf-Astoria

Study of Contamination of Surgical Wounds Occurring at Operation. JOHN J. MURPHY, M.D., HELEN ELLIS, B.A., and HAROLD A. ZINTEL, M.D., F.A.C.S., Philadelphia, Pennsylvania. Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania.

Further Studies in Evaluation of Preoperative Skin Preparation. HELEN ELLIS, B.A., JOHN J. MURPHY, M.D., and HAROLD A. ZINTEL, M.D., F.A.C.S., Philadelphia, Pennsylvania. Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania.

Experimental Use of Collagenase in Infected Wounds. S. C. MAY, M.D., SIDNEY E. ZIFFREN, M.D., F.A.C.S., R. E. KALLIO, Ph.D., and A. D. LARSON, M.S., Iowa City, Iowa. Departments of Surgery and Bacteriology, State University of Iowa College of Medicine.

Spontaneous Infections Following Thermal and Irradiation Injuries. W. A. ALTEMEIER, M.D., F.A.C.S., ALFRED KEIRLE, M.D., THOMAS LARGEN, M.D., and CHARLES BARRETT, M.D., Cincinnati, Ohio. Department of Surgery, University of Cincinnati College of Medicine.

Effect of Total Body Irradiation on Wound Closure. MICHAEL RADAKOVICH, M.D., ARTHUR M. DUTTON, Ph.D., and JOHN A. SCHILLING, M.D., F.A.C.S., Rochester, New York. University of Rochester School of Medicine and Dentistry, and Strong Memorial Hospital.

Role of Through-and-Through Tension Sutures in Healing of Wounds. J. H. O. MERTZ, M.D., H. C. MOSS, M.D., and HARRIS B. SHUMACKER, JR., M.D., F.A.C.S., Indianapolis, Indiana. Department of Surgery, Indiana University Medical Center.

Electrolyte Changes in Healing Wounds. JOHN A. SCHILLING, M.D., F.A.C.S., S. J. KUYKENDALL, M.D., T. J. WHELAN, JR., M.D., A. B. MCCOORD, Ph.D., and S. W. CLAUSEN, M.D., Rochester. New York. Department of Surgery, University of Rochester School of Medicine and Dentistry, and Strong Memorial Hospital.

Evaluation of Sympathetic Activity, Using Standard Equipment. LOUIS W. LEWIS, M.D., McKinney, Texas. Veterans Administration Hospital.

Effect of Altitude on E. Coli Peritonitis in Mice. WILLIAM H. HARRIDGE, M.D., and ALBERT J. NOVOTNY, M.D., Chicago, Illinois. Department of Surgery, University of Illinois College of Medicine.

Experimental Endogenous Peritonitis in Rat Response to Antibiotic Therapy. EDWIN J. PULASKI, M.D., D.Sc. (Surg.), F.A.C.S., (Lt. Col., M.C., USA), RUSSELL BRAME, B.S., HOWARD E. NOYES, M.S., Washington, D. C. Division of Surgery, Army Medical Service Graduate School, Walter Reed Army Medical Center, and Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas.

Injuries to Visceral Peritoneum: an Experimental Study. JEROME M. RINI, M.D., and RICHARD W. ZOLLINGER, M.D., F.A.C.S., Columbus, Ohio. Department of Surgery and Surgical Laboratories, Ohio State University College of Medicine.

Pancreatic Secretion of Antibiotics and Sulfadiazine in Man. JOHN M. HOWARD, M.D., (Capt., M.C., USA), EDWIN J. PULASKI, M.D., D.Sc. (Surg.), F.A.C.S., (Lt. Col., M.C., USA), MATTHEW J. FUSSILO, B.S., Washington, D. C. Division of Surgery, Army Medical Service Graduate School, Walter Reed Army Medical Center.

Staphylococcus Aureus Simultaneously Resistant to Penicillin and Broad Spectrum Antibiotics. MATTHEW J. FUSSILO, B.S., EDWIN J. PULASKI, M.D., D.Sc. (Surg.), F.A.C.S., (Lt. Col., M.C., USA), ERIC REISS, M.D., (Capt. M.C., USA), DWIGHT KUHNS, M.D., (Col., M.C., USA), Washington, D. C. Walter Reed Army Medical Center, Washington, and Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas.

Heart and Blood Circulation

Tuesday, 2:00–5:00 p.m., Sert Room, Waldorf-Astoria

Experimental Closure of Atrial Septal Defects; Technique of an "Atrial Well" Operation. ROBERT E. GROSS, M.D., F.A.C.S., A. A. POMERANZ, E. WATKINS, JR., and E. I. GOLDSMITH, Boston, Massachusetts. Harvard Medical School and The Children's Hospital.

Cardiovascular Responses to Partial or Complete Occlusion of the Aorta in Man During Operation. HUGH F. FITZPATRICK, M.D., ENZO KRAHL, M.D., SANFORD G. WEISSMAN, M.D., RALPH A. DETERLING, JR., M.D., F.A.C.S., ARTHUR H. BLAKEMORE, M.D., and GEORGE H. HUMPHREYS, II, M.D., F.A.C.S., New York, New York. Department of Surgery, Presbyterian Hospital, New York, and College of Physicians and Surgeons, Columbia University.

A Study of the Physiological Effects of a New Aortic Shunt. CHARLES A. HUBAY, M.D., ROBERT J. IZANT, M.D., and WILLIAM D. HOLDEN, M.D., Cleveland, Ohio. Department of Surgery, Western Reserve University School of Medicine, University Hospitals of Cleveland.

Factors Influencing Survival in Experimental Cardiac Tamponade. DENTON A. COOLEY, M.D., H. LEROY BROCKMAN, M.D., R. A. HUGGINS, Ph.D., Houston, Texas. Departments of Surgery and Pharmacology, Baylor University College of Medicine.

The Experimental Augmentation of Coronary Blood Flow. ADRIAN KANTROWITZ, M.D., and ARTHUR KANTROWITZ, Ph.D., Cleveland, Ohio. Department of Physiology, Western Reserve University School of Medicine, Graduate School of Aeronautical Engineering, Cornell University, Ithaca, New York.

Recovery of the Dog's Heart after Varying Periods of Acute Ischemia. SIGMUND A. WESOLOWSKI, M.D., JOHN H. FISHER, M.D., JOHN F. FENNESSEY, M.D., RICARDO CUBILES, M.D., and C. STUART WELCH, M.D., Boston. Pratt Diagnostic Clinic, New England Center Hospital.

The Effect of Repetitive Electrical Stimuli upon Heart Action. CHESTER E. HERROD, M.D., RUSSEL H. LEE, M.D., WALTER H. GOGGANS, M.D., ROLLIN K. MCCOMBS, M.A., and FRANK L. GERBODE, M.D., F.A.C.S., San Francisco, California. Stanford University School of Medicine.

Effect of *p*H Changes on Vagal Stimulation of the Heart. GILBERT S. CAMPBELL, FRANCIS J. HADDY, and E. B. BROWN, JR., Minneapolis. Departments of Surgery and Physiology, University of Minnesota Medical School.

Postural Variations in Peripheral Blood Flow in Venous and Arterial Vascular Diseases as Measured by the Electrical Impedance Plethysmograph. WILLIAM RILEY BOSIEN, M.D., MARIAN M. KREIDER, M.D., and JAN NYBOER, M.D., Hanover, New Hampshire. Departments of Physiological Sciences and Surgery, Dartmouth Medical School, Hanover, New Hampshire, and Surgical Service, Veterans Administration Hospital, White River Junction, Vermont.

Obstruction of the Inferior Vena Cava above the Renal Veins. CHARLES B. RIPSTEIN, M.D., F.A.C.S., F.R.C.S., DIRAN SEROPIAN, M.D., and ARTHUR LEVINE, M.D., New York, New York. Department of Surgery, Maimonides Hospital of Brooklyn, State University of New York College of Medicine.

Cardiodynamics of Experimental Infundibular (Pulmonary) Stenosis. JOHN F. ALDEN, JR., M.D., FRANCIS J. HADDY, WAYNE L. ADAMS, M.D., and IVAN D. BARONOFSKY, M.D., Minneapolis, Minnesota. Departments of Surgery and Physiology of the University of Minnesota, Ancker Hospital affiliation, St. Paul.

Further Experiences with the Technique of Producing Mitral Stenosis of Controlled Degree. ROBERT G. ELLISON, M.D., ROBERT C. MAJOR, M.D., F.A.C.S., RAYMOND W. PICKERING, M.D., and WILLIAM F. HAMILTON, M.D., Augusta, Georgia. Departments of Surgery and Physiology, Medical College of Georgia.

Circulatory Adjustments Observed During Mitral Valve Surgery. FERDINAND F. MCALLISTER, HUGH F. FITZPATRICK, M.D., SAMUEL R. POWERS, JR., EMANUEL M. PAPPER, M.D., and ENZO KRAHL, M.D., New York, New York. From the Departments of Surgery and Anesthesiology, Columbia University, College of Physicians and Surgeons and the Presbyterian Hospital.

Gynecology, Obstetrics, and Anesthesia

Tuesday, 2:00—5:00 p.m., Peacock Lounge, Waldorf-Astoria

Effect of Interstitial Cell Stimulating Hormone upon Uptake of P^{32} in Rat Prostate. MELVIN L. TAYMOR, M.D., JANET MCARTHUR, M.D., and FRANCIS INGERSOLL, M.D., F.A.C.S., Boston, Massachusetts. Departments of Gynecology of Massachusetts General and Massachusetts Memorial Hospitals.

Evaluation of Fluorescent Staining for Detection of Cancer Cells in Vaginal Smears. COY L. LAY, M.D., LAWRENCE M. RANDALL, M.D., and MALCOLM B. DOCKERTY, M.D., Rochester, Minnesota. Mayo Foundation for Medical Education and Research.

Complications Following Radiation Therapy of Cervical Malignancy. GEORGE L. HOFFMAN, JR., M.D., GEORGE C. LEWIS, JR., M.D., RICHARD H. CHAMBERLAIN, M.D., Philadelphia, Pennsylvania. Departments of Obstetrics and Gynecology, and Radiology, Hospital of the University of Pennsylvania.

Enhanced Effectiveness of Radiotherapy in Cancer of Uterine Cervix. JOHN B. GRAHAM, M.D., F.A.C.S., and RUTH M. GRAHAM, M.D., Boston, Massachusetts. Vincent Memorial Hospital.

Use of Corticotropin in Rh Isoimmunization. R. C. CHRISTENSEN, M.D., R. R. MARGULIS, M.D., and HARRY L. STEWART, JR., M.D., F.A.C.S., Detroit, Michigan. Department of Obstetrics and Gynecology, Henry Ford Hospital.

Use of Cortisone in Pelvic Cellulitis. NEILSON M. MATHEWS, JR., M.D., CONRAD G. COLLINS, M.S., M.D., F.A.C.S., New Orleans, Louisiana. Tulane Unit of Charity Hospital, Tulane University of Louisiana School of Medicine.

Air and Amniotic Fluid Embolism in Obstetrics. JOSEPH P. GRIFFON, M.D., and ISADORE DYER, M.D., F.A.C.S., New Orleans, Louisiana. Tulane Unit of Charity Hospital, Tulane University of Louisiana School of Medicine.

Recent Advances in Management of Vaginitis. WARREN R. LANG, M.D., A. E. RAKOFF, M.D., and R. SOSNOWSKI, M.D., Philadelphia, Pennsylvania.

Double Uterus and Repeated Abortion; Indication for Surgical Repair. HOWARD W. JONES, JR., M.D., Baltimore, Maryland. Department of Gynecology, Johns Hopkins University Medical School.

Hemodynamic Changes During Thiopental Anesthesia in Man. EUGENE H. CONNER, M.D., ARTHUR VALK, JR., M.D., and HENRY L. PRICE, M.D., Philadelphia, Pennsylvania. Department of Anesthesiology, Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Clinical and Laboratory Observations of Use of Xenon for Anesthesia. STUART C. CULLEN, M.D., and CHARLES B. PITTINGER, M.D., Iowa City, Iowa. Division of Anesthesiology, State University of Iowa College of Medicine.

Insulin as a Possible Pentothal Sodium Antagonist. W. P. EDER, M.D., MALCOLM D. MCCAMPBELL, M.D., Minneapolis, Minnesota. Department of Surgery, Minneapolis General Hospital and Department of Surgery, University of Minnesota Medical School.

Lungs and Liver

Wednesday, 9:00 a.m.—12:00, Sert Room, Waldorf-Astoria

Changes in Respiratory Dynamics Following Tracheotomy. WILLIAM S. BLAKEMORE, M.D., Philadelphia, Pennsylvania. Harrison Department of Surgical Research, Department of Physiology, Graduate School of Medicine, University of Pennsylvania.

Preliminary Observations on Homologous Lung Transplants in Dogs. CREIGHTON A. HARDIN, M.D., C. FREDERICK KITTLE, M.D., and PAUL W. SCHAFER, M.D., F.A.C.S., Kansas City, Kansas. Department of Surgery, University of Kansas Medical Center.

Experimental Lung Transplantation. WILFORD B. NEPTUNE, M.D., HECTOR REDONDO, M.D., and CHARLES P. BAILEY, M.D., F.A.C.S., Philadelphia, Pennsylvania. Hahnemann Medical College and Hospital of Philadelphia.

Oxymetry During Thoracic Operations. JAMES J. FINNERTY, M.D., and ERIC CARLENS, Medical Director, New York. Third (New York University) Surgical Division, Bellevue Hospital. The Sabbatsbergs Sjukhus, Stockholm, Sweden.

Treatment of Pulmonary Edema with Portable Mechanical Respirators. DAVID G. GREENE, M.D., and JOSEPH T. DAMERON, M.D., Buffalo, New York. Buffalo General Hospital.

The Effect of Multiple Pulmonary Emboli on Abnormal Cardiopulmonary States. M. HARA, M.D., W. MURRY, M.D., and JAMES WALT, M.D., Little Rock, Arkansas. Department of Surgery, University of Arkansas School of Medicine.

The Treatment of Pulmonary Insufficiency by Reduction of Body Oxygen Requirements. CHARLES E. O'BRIEN, M.D., and CLYDE E. RUSH, M.D., Ann Arbor, Michigan. University of Michigan Medical School.

A Study of Liver Failure in the Dog Due to Biliary Obstruction. CHARLES A. MACGREGOR, M.D., Boston, Massachusetts. Department of Surgery, Peter Bent Brigham Hospital, Laboratory for Surgical Research, Harvard Medical School.

The Pathogenesis of Ascites in Cirrhosis of the Liver. JOHN L. MADDEN, M.D., F.A.C.S., JOHN M. LORE, M.D., and FRANK P. GEROLD, M.D., New York, New York. Department of Surgery, Surgical Research Laboratory, St. Clare's Hospital.

Effect of Partial Hepatectomy, Protein Realimentation, Bile, 1 Per Cent Methionine Diet, Ether, and Chloroform, on Biliary Secretion in the Rat. BERNARD FISHER, M.D., JOAN W. ZERBE, and HARRY M. VARS, Ph.D., Philadelphia, Pennsylvania. Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Alterations in Common Bile Duct Stricture Formation by Proximal Decompression. WILLIAM H. HARRIDGE, M.D., Chicago, Illinois. Department of Surgery, University of Illinois College of Medicine.

Studies in the Source and Variations of Amylase in Animal and Human Bile. R. L. ESTRADA, M.D., F.R.C.S. (C), Montreal, Canada. Department of Experimental Surgery, McGill University.

Shock

Wednesday, 9:00 a.m.—12:00, Empire Room, Waldorf-Astoria

Modified Fluid Gelatin as a Plasma Volume Expander in Hemorrhagic Hypotensive Dogs. WILLIAM M. PARKINS, Ph.D., JOSEPH H. PERLMUTT, Ph.D., and HARRY M. VARS, Ph.D., Philadelphia. The Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Some Physiological Effects Following a Dextran Infusion in Normal Subjects. WILLIAM METCALF, M.D., F.A.C.S., and LOUIS M. ROUSSELOT, M.D., New York, New York. Departments of Surgery, St. Vincent's Hospital and New York University College of Medicine. Work performed under contract with the Office of Medical Research and Development, United States Army.

Evaluation of Plasma Substitutes in Wound Shock: Polyvinylpyrrolidone. B. W. HAYNES, JR., M.D., E. STANLEY CRAWFORD, M.D., J. HERMAN MAHAFFEY, M.D., F.A.C.S., and MICHAEL E. DEBAKEY, M.D., F.A.C.S., Houston, Texas. Department of Surgery, Baylor University College of Medicine.

An Evaluation of Methods for Comparing Plasma Volume Expanders in Humans. HAROLD G. BARKER, M.D., JOHN D. ELDER, M.D., JAMES M. WALKER, M.D., and HARRY M. VARS, Ph.D., Philadelphia. The Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Clinical Experience with Fluid Gelatin. JOHN W. THOMAS, M.D., and JONATHON E. RHOADS, M.D., F.A.C.S., Philadelphia, Pennsylvania. The Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

The Effects on Pulmonary Red Blood Cell and Plasma Volumes of Hemorrhage and Replacement Therapy in Dogs With and Without Sodium Depletion. BEN J. WILSON, M.D., MORRIS J. FOGELMAN, M.D., and ALLEN F. REID, Ph.D., Dallas, Texas. The Departments of Surgery and Biophysics, Southwestern Medical School of The University of Texas.

Changes in the Pulmonary Circulation During Hemorrhagic Shock and Resuscitation. HARRY L. ROUNTHWAITE, M.D., HARRY J. SCOTT, M.D., F.R.C.S.(C), and FRASER N. GURD, M.D., F.R.C.S.(C), F.A.C.S., Montreal, Canada. Department of Experimental Surgery, McGill University.

Factors Concerned in the Production of Renal Disease Following Transfusion of Mismatched Blood. C. BARBER MUELLER, M.D., BEN EISEMAN, M.D., A. D. MASON, M.D., F.A.C.S., and P. T. NORMAN, M.D., St. Louis, Missouri. Department of Surgery, Washington University School of Medicine.

Hemolytic Transfusion Reactions During Surgery. MORRIS J. FOGELMAN, M.D., K. I. ROBINOWITZ, and W. F. STAPP, M.D., Dallas, Texas. Departments of Surgery and Pathology, Southwestern Medical School of The University of Texas.

Permeability of the Intestine to Bacterial Toxins in Hemorrhagic Shock. RUSSELL M. NELSON, M.D. (1st Lt., M.C., USA), and HOWARD E. NOYES, M.S., Washington, D. C. Army Medical Service Graduate School, Walter Reed Army Medical Center.

Studies on the Recovery and in Vivo Survival of Animal and Human Red Cells After Prolonged Preservation at Subzero Temperatures. IVAN W. BROWN, JR., M.D., Durham, North Carolina. Duke University School of Medicine.

Portal Hypertension, Liver, Pancreas, and Blood Coagulation

Wednesday, 2:00—5:00 p.m., Sert Room, Waldorf-Astoria

Effect of Occlusion of Vessels Entering the Liver on Hepatic Venous Pressures in Normal and Cirrhotic Dogs. JOHN H. GRINDLAY, M.D., F.A.C.S., RICHARD THORS, M.D., JESSIE L. BOLLMAN, M.D., Rochester, Minnesota. Division of Experimental Medicine, Mayo Foundation.

Observations on the Inconstancy of Portal Hypertension in Hepatic Cirrhosis. WILLIAM S. DYE, M.D., HUGH BENNETT, M.D., LYLE BAKER, M.D., ORMAND C. JULIAN, M.D., F.A.C.S., Chicago, Illinois. Department of Surgery, University of Illinois College of Medicine, Chicago, Illinois, and the Departments of Medicine and Surgery of the Veterans Administration Hospital, Hines, Illinois.

Effect of Ligation of the Hepatic Artery on Oxygen Content of Blood from the Hepatic Vein in Dogs. RICHARD THORS, M.D., JOHN H. GRINDLAY, M.D., F.A.C.S., and JESSE L. BOLLMAN, M.D., Rochester, Minnesota. Mayo Foundation for Medical Education and Research.

Graded Hepatic Arterial Ligations in Experimental Ascites. HAROLD LAUFMAN, M.D., Ph.D., F.A.C.S., ALFRED ROSS, M.D., VICTOR M. BERNHARD, M.D., ROBERT V. BOURDEAU, M.D., and WALTER E. FURR, JR., M.S., M.D., Chicago. Department of Surgery, Northwestern University Medical School.

Ligation of the Hepatic and Splenic Arteries in the Treatment of Cirrhosis of the Liver. JOHN L. MADDEN, M.D., F.A.C.S., New York, New York. Department of Surgery, St. Clare's Hospital.

Experimental Hepatic Coma. A. M. RAPPAPORT, Z. J. BOROWY, and W. N. LOTTO, M.D., Toronto, Canada. Department of Physiology, University of Toronto, Faculty of Medicine.

Experimental Ascites; the Influence of Transplantation of the Kidneys to the Neck. T. J. WHELAN, M.D., A. B. MCCOORD, and J. A. SCHILLING, M.D., F.A.C.S., Rochester, New York. Department of Surgery, University of Rochester School of Medicine and Dentistry, and Strong Memorial Hospital.

Production of Recurrent Acute Pancreatitis in Dogs. JOHN H. WAITE, M.D., Columbus, Ohio. Department of Surgery and Surgical Laboratories, Ohio State University, College of Medicine.

Observation of Changes in Blood Coagulability in Patients with Pancreatic Disease. WILLIAM W. SHINGLETON, M.D., WILLIAM G. ANLYAN, M.D., KARLEEN C. NEILL, M.D., THOMAS D. VANCE, Durham, North Carolina, Department of Surgery, Duke University School of Medicine.

The Effects of Intravenous Trypsin (Tryptar) upon Thrombophlebitis and the Acute Inflammatory Reaction. IRVING INNERFIELD, M.D., ALFRED W. SCHWARTZ, M.D., ALFRED A. ANGRIST, M.D., and WILFRED F. RUGGIERO, M.D., F.A.C.S., New York, New York. New York Medical College, Flower and Fifth Avenue Hospitals, Research Unit, Metropolitan Hospital, Jewish Memorial Hospital.

The Effect of Intravenous Administration of Activated Human Plasma Euglobulin in Dogs. DONALD J. GLOTZER, M.D., and J. GARROTT ALLEN, M.D., F.A.C.S., Chicago. Department of Surgery, The University of Chicago School of Medicine.

Evaluation of the Various Methods of Administration of Heparin. CURTIS P. ARTZ, M.D., M.S. (Surg.), (Major, M.C.), and WILLIAM H. AMSPACHER, M.D. (Col., M.C.), Fort Sam Houston, Texas. Surgical Research Unit, Brooke Army Hospital, Fort Sam Houston, Texas.

Postoperative Thromboembolism—II, Relationship of True Antithrombin to the Kay Test and Evaluation of Alpha Tocopherol-Calcium Gluconate Prophylaxis. WILLIAM W. COONE, M.D., ROBERT M. WHITROCK, M.D., and KENT D. MILLER, Ann Arbor, Michigan. Department of Surgery, University of Michigan Medical School.

Orthopedic Surgery

Wednesday, 2:00—5:00 p.m., Peacock Lounge, Waldorf-Astoria

Study of Vascularization of Bone Transplants by Means of Transparent Chamber and Microscopic Sections. CLIFFORD L. KIEHN, M.D., F.A.C.S., DONALD M. GLOVER, M.D., F.A.C.S., and JAMES GUTENTAG, B.S., Cleveland, Ohio. Department of Surgery, Western Reserve University School of Medicine.

Effect of Microwaves on Healing of Experimentally Produced Fractures in Rabbits. EDWARD D. HENDERSON, M.D., Rochester, Minnesota. Mayo Foundation.

Fanconi Syndrome; Report of a Case Showing Response to Vitamin D. E. C. FRONING, M.D., GENEVIEVE STEARNS, Ph.D., M. BONFIGLIO, M.D., Iowa City, Iowa. State University of Iowa College of Medicine.

Early Surgical Biopsy of Vertebral Body Lesions; a Diagnostic Necessity. R. W. JOHNSON, JR., M.D., J. W. HILLMAN, M.D., and W. O. SOUTHWICK, M.D., Baltimore, Maryland. Johns Hopkins Hospital.

Experiments In Leg Lengthening by Stasis; a Preliminary Report. WILLIAM J. HUTCHISON, M.D., and B. D. BURDEAUX, M.D., McKinney and Dallas, Texas. Veterans Administration Hospitals.

Study of Stress in Relation to Healing of Bone. JAMES A. NICHOLAS, M.D., PHILIP D. WILSON, M.D., F.A.C.S., and CHARLES UMBERGER, Ph.D., New York, New York. Columbia University College of Physicians and Surgeons.

Bone Changes Following Ureterosigmoidostomy. MARY S. SHERMAN, M.D., F.A.C.S., Chicago, Illinois. Division of Orthopedic Surgery. Department of Surgery, University of Chicago School of Medicine.

Mechanism and Clinical Correction of Trendelenburg Gait. CHARLES O. BECHTOL, M.D., Oakland, California.

Aspiration Biopsy of the Spine. CHARLES J. FRANKEL, M.D., Charlottesville, Virginia. Department of Orthopedic Surgery, University of Virginia Hospital.

Effects of Compression on Healing of Fractures. J. M. MCINTYRE, M.D., and JAMES G. SHANNON, M.D., F.A.C.S., Montreal, Canada. Department of Experimental Surgery, McGill University Faculty of Medicine.

Experimental Study of the Use of Gelfoam, With and Without Calcium Salts, to Fill Defects in Bone. FRED C. REYNOLDS, M.D., F.A.C.S., and LEE T. FORD, M.D., St. Louis, Missouri. Washington University School of Medicine.

Histologic Studies Following Exposure of Bone to Ultrasound; an Experimental Investigation. L. F. BENDER, M.D., JOSEPH M. JANES, M.D., F.A.C.S., and J. F. HERRICK, Ph.D., Rochester, Minnesota. Mayo Clinic.

Tendon Transplantation Through Artificially Created Tendon Sheaths. JOSEPH E. MILGRAM, M.D., F.A.C.S., Brooklyn, New York.

Influence of Hyaluronidase on Experimental Bone Repair and Ectopic Bone Formation. J. J. GARTLAND, M.D., and W. R. MACAUSLAND, JR., M.D., New York. Presbyterian Hospital, Columbia-Presbyterian Medical Center, New York Orthopedic Hospital.

Metals and Plastics in Orthopedic Surgery and General Surgery. J. W. BLUNT, JR., M.D., STEPHEN S. HUDACK, M.D., F.A.C.S., and CLAY RAY MURRAY (Deceased), M.D., F.A.C.S., New York, New York. Presbyterian Hospital, Columbia-Presbyterian Medical Center.

Water Balance and the Electrolytes

Thursday, 9:00—12:00, Sert Room, Waldorf-Astoria

The Effect of Thyroid Disease on Water Turnover Rate in the Human Subject. HAROLD B. HALEY, M.D., ISIDORE S. EDELMAN, M.D., DONALD S. FREDERICKSON, M.D., FRANCIS D. MOORE, M.D., F.A.C.S.,

Boston, Massachusetts. Department of Surgery, Peter Bent Brigham Hospital, Laboratory for Surgical Research, Harvard Medical School.

Rate of Fat Oxidation in the Surgical Patient. FRANCIS D. MOORE, M.D., F.A.C.S., LAURA BROOKS, M.D., and ISIDORE S. EDELMAN, M.D., Boston, Massachusetts. Department of Surgery, Peter Bent Brigham Hospital, Laboratory for Surgical Research, Harvard Medical School.

A Comparison of the Effect of Operation on Glucose and Fructose Metabolism. WILLIAM R. DRUCKER, M.D., MAX MILLER, M.D., JAMES CRAIG, M.D., WILLIAM MCKENDREE JEFFERIES, M.D., STANLEY LEVEY, Ph.D., and WILLIAM E. ABBOTT, M.D., F.A.C.S., Cleveland, Ohio. Departments of Surgery and Medicine, Western Reserve University School of Medicine, University Hospitals of Cleveland.

Extracellular Fluid Volume and Electrolytes in Thoracic Surgical Patients. JOHN Y. TEMPLETON, III, M.D., Philadelphia, Pennsylvania. Jefferson Medical College of Philadelphia.

Distortion of the Normal Fluid and Electrolyte Pattern Following Extravascular Clysis. ROBERT C. FOREMAN, M.D., FRANK A. CEBUL, M.D., STANLEY LEVEY, Ph.D., and WILLIAM E. ABBOTT, M.D., F.A.C.S., Cleveland, Ohio. Department of Surgery, Western Reserve University School of Medicine, University Hospitals of Cleveland.

Adrenal Function in Surgical Patients; a Comparison of Results in Healthy and in Chronically Ill, Debilitated Patients. F. HENRY ELLIS, JR., M.D., HAROLD L. MASON, Ph.D., and JAMES T. PRIESTLEY, M.D., F.A.C.S., Rochester, Minnesota. Mayo Foundation for Medical Education and Research.

Changes in the Electrophoretic Pattern of the Plasma Proteins as Induced by Surgery. R. R. MARGULIS, M.D., MARGARET E. WISEMAN, and ELSIE Z. MOYER. Detroit, Michigan. Department of Obstetrics and Gynecology, Henry Ford Hospital, Research Laboratory of the Children's Fund of Michigan.

Utilization of a Subcaloric Amino Acid Mixture Given Intravenously to the Extremely Malnourished Patient. E. J. CONRAD, M.D., and M.D. PAREIRA, M.D., F.A.C.S., St. Louis, Missouri. Department of Surgery, Washington University School of Medicine.

Evaluation of a "Balanced" Amino Acid Solution for Parenteral Use. TILDEN C. EVERSON, M.D., JOHN F. LAWS, M.D., and EVERETT T. HOPPE, Chicago, Illinois. Department of Surgery, University of Illinois College of Medicine.

Comparison of Effects of Intravenous Administration of 5 Per Cent Glucose and 10 Per Cent Invert Sugar on Water and Electrolyte Excretion. PAUL E. HODGSON, M.D., VIVIAN IOB, Ph.D., and WILLIAM W. COON, M.D., Ann Arbor, Michigan. University of Michigan Medical School.

The Utilization of Invert Sugar and Glucose Following Intravenous Administration to Postoperative Patients. OTTO ROSENTHAL, M.D., WILLIAM C. STAINBACK, M.D., and JONATHAN E. RHOADS, M.D., F.A.C.S., Philadelphia, Pennsylvania. Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Classification of Edema and Dehydration in Surgical Patients as Determined by Muscle Biopsies. IRVING M. ARIEL, M.D., ARNOLD J. KREMEN, M.D., and ESTHER FRIER, B.S., Minneapolis, Minnesota. Department of Surgery, Clinical Laboratory Service, University of Minnesota.

Neurosurgery

Thursday, 9:00 a.m.—12:00, Empire Room, Waldorf-Astoria

A Clinical and Experimental Determination of Pressure Within the Carotid Arteries. WILLIAM J. GERMAN, M.D., and SAMUEL P. W. BLACK, M.D., New Haven, Connecticut. From the Department of Surgery, Yale University, New Haven, Connecticut.

Observations on the Course of Cerebrospinal Fluid Pressure as Influenced by Certain Anesthetic Agents. ERNEST J. PENKA, M.D., Cincinnati, Ohio. Laboratory of Neurophysiology, Division of Neurosurgery, Department of Surgery, Cincinnati General Hospital, University of Cincinnati College of Medicine.

The Significance of Cervical and Intracranial Arterial Pressures in the Treatment of Aneurysms and Neoplasms. LOUIS BAKAY, M.D., and WILLIAM H. SWEET, Harvard Medical School.

Investigation on Blood-Brain and Blood-Liquor Barrier Using Radioisotope Techniques. GERALD L. HAINES, M.D., Minneapolis, Minnesota. Department of Surgery, Division of Neurosurgery, University of Minnesota Medical School.

Radioactive Water; a New Tool for Study of Cerebrospinal Fluid. BERTRAM SELVERSTONE, M.D., P. BENDA, C. V. ROBINSON, Boston, Massachusetts. Tufts College Medical School.

Recent Advances in Treatment of Communicating Hydrocephalus. EUGENE B. SPITZ, M. D., and C. EVERETT KOOP, M. D., Sc.D.(Med.), F.A.C.S., Philadelphia, Pennsylvania. Children's Hospital of Philadelphia, Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Effect of Previous Sensory Loss on Appearance of Herpes Simplex Following Trigeminal Sensory Root Section. CHARLES A. CARTON, M. D., New York, New York. Neurological Institute, Presbyterian Hospital.

Fibroblastic Stimulation and Thickening of Rabbit Femoral Artery Adventitia by Marine Varnish, Krylon,

and Latex; a Laboratory Study Related to Surgery of Congenital Cerebral Aneurysm. BARNES WOODHALL, M.D., F.A.C.S., and JAMES GOLDEN, M.D., Durham, North Carolina. Duke University School of Medicine.

Effect of Interruption of Hypothalamo-hypophyseal Pathways in the Dog. GEORGE J. WITTENSTEIN, M.D., and ERNST A. SCHARRER, M.D., Denver, Colorado. Departments of Surgery and Anatomy, University of Colorado School of Medicine.

Blood Supply of Optic Chiasm and Intracranial Portion of Optic Nerves; Study of Arterial Injections with Latex in the Macacus Rhesus Monkey and Human. DONALD H. LYLE, M.D., F.A.C.S., CURWOOD R. HUNTER, M.D., and FRANK H. MAYFIELD, M.D., F.A.C.S., Cincinnati, Ohio.

Hypothalamic and Brain Stem Lesions in Hypertensive Dogs. K. M. BROWNE, M.D., and J. D. MCQUEEN, M.D., Baltimore. Division of Neurological Surgery, Johns Hopkins University School of Medicine.

A Method for Reduction of Peripheral Vascular Bed of an Extremity Without Occlusion of Major Arterial Channels. ROBERT B. KING, M.D., and J. LOUIS SCHRICKER, M.D., St. Louis, Missouri. Department of Surgery (Division of Neurological Surgery), Washington University School of Medicine.

Relative Accuracy of Electroencephalography, Air Studies and Angiography in a Series of Two Hundred Mass Lesions. FRANCIS A. MARTIN, M.D., JOHN E. WEBSTER, M.D., F.A.C.S., and E. S. GURDJIAN, M.D., F.A.C.S., Detroit, Michigan.

Urology

Thursday, 1:30—3:15 p.m., Sert Room, Waldorf-Astoria

Determination of Individual Renal Function. TOM E. NESBITT, M.D., Ann Arbor, Michigan. University of Michigan Medical School.

Evaluation of Aortography as a Diagnostic Aid. CHARLES D. CREEVY, M.D., R. W. KRUMBACH, M.D., and M. J. FEENEY, M.D., Minneapolis, Minnesota. Division of Urology, Department of Surgery, University of Minnesota Medical School.

An Experimental Study of Function of Homologous and Heterologous Kidneys Implanted into the Systemic Circulation of Uremic Dog. ALLEN JOHNSON, M.D., PHILIP GOLDMAN, M.D., and HORACE J. MCCORKLE, M.D., F.A.C.S., San Francisco, California. Experimental Surgery Laboratories, University of California School of Medicine.

Homologous Transplantation of Kidneys in the Human. DAVID M. HUME, M.D., JOHN P. MERRILL, M.D., and BENJAMIN F. MILLER, M.D., Boston, Massachusetts. Department of Surgery and Medicine, Peter Bent Brigham Hospital, Harvard Medical School.

A Study of Experimental Renal Tubular Damage in The Dog. ROBERT E. DESAUTELS, M.D., JOHN T. FINKENSTAEDT, M.D., and CARL W. WALTER, M.D. Boston, Massachusetts. Department of Surgery, Peter Bent Brigham Hospital, Laboratory for Surgical Research, Harvard Medical School.

A New Concept of Vesical Innervation and its Clinical Application. LOWRAIN E. MCCREA, M.D., F.A.C.S., Philadelphia, Pennsylvania.

Experimental Studies on Electrolyte Exchanges in the Dog's Colon. ANGELO D'AGOSTINO, M.D., Boston, Massachusetts. New England Center Hospital, Ziskind Research Laboratories Pratt Diagnostic Clinic, Tufts College Medical School.

Observations on Use of Cecum as Substitute Urinary Bladder. JAMES G. M. HARPER, M.D., MICHAEL H. BERMAN, M.D., ARTHUR D. HERTZBERG, M.D., FRED LERMAN, M.D., and HERBERT BRENDLER, M.D., F.A.C.S., New York, New York. Urology Service, Veterans Administration Hospital, (Bronx), Department of Urology, New York University Post-Graduate Medical School.

Preliminary Report on Experimental Use of Polyethylene Tubing in Reconstitution of the Ureter. CHARLES J. ROBSON, M.D., F.R.C.S. (C), Toronto, Ontario, Canada. Department of Surgery, University of Toronto, Faculty of Medicine.

Furadantin in Urinary Tract Infections Due to Bacillus Proteus. CHARLES E. FRIEDGOOD, M.D., and ANTHONY DANZA, M.D., New York, New York. Department of Surgery, Maimonides Hospital of Brooklyn, State University of New York College of Medicine.

Plastic Surgery

Thursday, 1:30—3:15 p.m., Peacock Lounge, Waldorf-Astoria

Experimental Production of Congenital Defects, with Particular Reference to Cleft Palate. F. CLARKE FRASER, Ph.D., M.D., T. D. FAINSTAT, M.Sc., and H. KALTER, M.Sc., Montreal, Canada. McGill University Faculty of Medicine.

Syngenesiotransplantation in Parabiotic Rats. W. E. LANSCHE, M.D., and A. E. MCGINNIS, M.D., St. Louis, Missouri. Washington University School of Medicine.

Development of Circulation in Autogenous and Homologous Grafts of Skin and Observations on Effect of Hyaluronic Acid and Homologous Skin Filtrate. HERBERT CONWAY, M.D., F.A.C.S., RICHARD B. STARK, M.D., and DOYLE JOSLIN, Ph.D., M.D., New York, New York. New York Hospital and Cornell University Medical College.

Reconstruction of the Thumb. ERLE E. PEACOCK, JR., M.D., (Lt., M.C., USAR), Phoenixville, Pennsylvania. Hand Section, Valley Forge Army Hospital.

A Method for the Repair of Experimentally Produced Large Body Wall Defects in Dogs by Use of Flexible Polyethylene Plates. PHILIP GOLDMAN, M.D., ALLEN JOHNSON, M.D., and HORACE J. MCCORKLE, M.D., F.A.C.S., San Francisco, California. Experimental Surgery Laboratories, University of California School of Medicine.

A Method for Determining Viability of Experimental Human Skin Grafts. JOHN H. TENERY, M.D., St. Louis. Division of Plastic Surgery, Department of Surgery, Washington University School of Medicine.

The Effects of Various Agents on Healing in Controlled Partial Thickness Skin Loss. PETER RANDALL, M.D., St. Louis, Missouri. Division of Plastic Surgery, Department of Surgery, Washington University School of Medicine.

Reconstructive Techniques to Facilitate Treatment of Head and Neck Cancer. MILTON T. EDGERTON, JR., M.D., Baltimore, Maryland. Department of Plastic and Maxillofacial Surgery, Johns Hopkins Hospital and Johns Hopkins University School of Medicine.

An Experimental Study of Preservation of Skin Grafts by Freeze-Drying Process. F. TALMADGE BUCHANAN, M.D., and EDWIN P. LEHMAN, M.D., F.A.C.S., Charlottesville, Virginia. Department of Surgery, University of Virginia Department of Medicine.

Thermal Injuries and Cancer

Friday, 9:00 a.m.—12:00, Sert Room, Waldorf-Astoria

Experiences with Oral High-Protein Feeding in Severe Burn Patients. TRUMAN G. BLOCKER, JR., M.D., F.A.C.S., VIRGINIA BLOCKER, M.D., S. R. LEWIS, M.D., and C. C. SNYDER, M.D., Galveston, Texas. Department of Plastic and Maxillofacial Surgery, University of Texas Medical Branch, Galveston.

Mechanism of Death in Burns; a Clinical Pathologic Study. THOMAS M. CLOUD, M.D., (Lt., M.C.), CURTIS P. ARTZ, M.D., M.S. (Surg.), (Maj., M.C.), and WILLIAM H. AMSPACHER, M.D., (Col., M.C.), Fort Sam Houston, Texas. Surgical Research Unit, Brooke Army Hospital, Fort Sam Houston, Texas.

Thermal Burns of the Respiratory Tract. CHARLES FINEBERG, M.D., BERNARD J. MILLER, M.D., and FRANK F. ALLBRITTEN, JR., M.D., F.A.C.S., Philadelphia. Jefferson Medical College of Philadelphia.

Studies on the Physiology, Biochemistry, and Therapy of Thermal Burns. CHARLES L. FOX, JR., M.D., SIGMUND LASKER, and JAMES M. WINFIELD, M.D., F.A.C.S., New York, New York. Department of Surgery, New York Medical College, Flower and Fifth Avenue Hospitals.

Tissue Chloride Changes in the Burned Rat. JAMES M. WALKER, M.D., MARIE T. BARNES, R.N., MORTON D. GLASSMAN, and AVIS R. WALKER, Philadelphia, Pennsylvania. Harrison Department of Surgical Research, University of Pennsylvania Schools of Medicine.

Effect of Rapid Thawing, Vasodilatation, and ACTH in Treatment of Acute Frostbite. MARTIN A. ENTIN, M.Sc., M.D., Montreal, Canada. Department of Experimental Surgery, McGill University Faculty of Medicine.

Redistribution of Potassium and Sodium in Experimental Frostbite. W. J. PIROZYNSKI, M.D., and D. R. WEBSTER, M.D., Ph.D., F.A.C.S., Montreal, Canada. Department of Experimental Surgery, McGill University Faculty of Medicine.

Abrasive Gastric Balloon Test for Detecting Gastric Cancer. FREDERICK G. PANICO, M.D., Baltimore. Baltimore City Hospitals, Department of Surgery, and University of Maryland School of Medicine.

Use of Insects in the Study of Cancer. WALTER J. BURDETTE, Ph.D., M.D., F.A.C.S., New Orleans, Louisiana. Department of Surgery, Louisiana State University School of Medicine.

Serum Aldolase and Its Relationship to Hormonal Control of Cancer. ROGER BAKER, DUNCAN GOVAN, and JAMES CASON, Chicago, Illinois. Department of Surgery (Division of Urology), University of Chicago, The School of Medicine of the Biological Sciences.

Effect of Thyrotropic Hormone and Propylthiouracil on Homologous Thyroid Transplants. JOSEPH T. DAMERON, M.D., Buffalo, New York. Buffalo General Hospital, and Department of Surgery, University of Buffalo School of Medicine.

Tobacco and Intraoral Cancer. GEORGE E. MOORE, M.D., L. L. BISSINGER, M.D., and E. ANDERSON, M.D., Minneapolis, Minnesota. Department of Surgery, University of Minnesota Medical School.

Radioactive Colloidal Gold in Treatment of Peritoneal and Pleural Effusions Due to Neoplasms. JOHN P. STORAASLI, M.D., FREDERICK J. BONTE, M.D., and HYMER L. FRIEDELL, M.D., Cleveland, Ohio. Department of Radiology, Western Reserve University School of Medicine and University Hospitals.

Effect of Local Lesions on Incidence of Breast Tumors Produced by 2-Acetylaminofluorene. ROBERT F. SCARF, M.D., RODERICK C. ROSS, M.Sc., M.D., and STANLEY C. SKORYNA, M.Sc., M.D., Montreal. Departments of Experimental Surgery and Pathology, McGill University Faculty of Medicine.

Thromboplastic Property of Neoplasms. EDWIN A. LAWRENCE, M.D., F.A.C.S., DONALD B. MOORE, M.D., and GEORGE I. BERNSTEIN, M.D., Indianapolis, Indiana. Department of Surgery, Indiana University Medical Center.

THE CLINICAL CONGRESS TECHNICAL EXHIBITION

The Clinical Congress Technical Exhibition provides an attractive method of displaying supplies and equipment designed for use in hospitals and surgeons' offices, such as surgical instruments, diagnostic and therapeutic apparatus, pharmaceuticals, medical literature, and other similar items. The 1952 Technical Exhibition will be located at the Waldorf-Astoria Hotel in New York. James S. Shannon, General Manager of The Franklin H. Martin Memorial Foundation, publishers of the official College journal, SURGERY, GYNECOLOGY AND OBSTETRICS, is Manager of the Technical Exhibition. The list of exhibitors follows:

Abbott Laboratories, North Chicago, Ill.
W. D. Allison Company, Indianapolis, Ind.
American Cystoscope Makers, Inc., New York
American Hospital Supply Corporation, Evanston, Ill.
American Ossacryl Company, New York
American Safety Razor Corp., Brooklyn
American Sterilizer Company, Erie, Pa.
Ames Company, Inc., Elkhart, Ind.
The Armour Laboratories, Chicago
Association of American University Presses, New York
Austenal Laboratories, Inc., New York
C. R. Bard, Inc., Summit, N. J.
Bard-Parker Company, Inc., Danbury, Conn.
Bauer and Black, Chicago
Rudolph Beaver, Waltham, Mass.
Becton, Dickinson and Company, Rutherford, N. J.
Julius Berbecker & Sons, Inc., New York
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Cameron Surgical Specialty Company, Chicago
S. H. Camp and Company, Jackson, Mich.
Wilmot Castle Company, Rochester, N. Y.
Chesebrough Mfg. Co., Cons'd., N. Y.
Gilbert Hyde Chick Co., Oakland, Calif.
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Ciba Pharmaceutical Products, Inc., Summit, N. J.
Clay-Adams Company, Inc., New York
The Coca-Cola Company, Atlanta
Cochrane Physicians' Supplies, Inc., New York
Codman & Shurtleff, Inc., Boston
Commercial Solvents Corporation, New York
Cutter Laboratories, Berkeley, Calif.
Davis & Geck, Inc., Brooklyn
J. A. Deknatel & Son, Inc., Queens Village, L. I., N.Y.
DePuy Manufacturing Co., Inc., Warsaw, Ind.
Desitin Chemical Company, Providence, R. I.
Duke Laboratories, Inc., Stamford, Conn.
E & J Manufacturing Co., Burbank, Calif.
J. H. Emerson Co., Cambridge, Mass.
Ethicon Suture Laboratories, Inc., New Brunswick
Harold L. Feighner & Company, Detroit
C. B. Fleet Co., Inc., Lynchburg, Va.
E. Fougera & Co., Inc., New York
Grune & Stratton, Inc., New York
Gudebrod Bros. Silk Co., Inc., New York
J. E. Hanger, Inc., Washington, D. C.
Paul B. Hoeber, Inc., New York
Hoffmann-La Roche, Inc., Nutley, N. J.
Johnson & Johnson, New Brunswick
Kansas City Assemblage Company, Kansas City, Mo.
Lea & Febiger, Philadelphia
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Eli Lilly and Company, Indianapolis
J. B. Lippincott Company, Philadelphia
The Macmillan Company, New York
Mead Johnson & Company, Evansville, Ind.
Medco Products Company, Tulsa
Medical Aids, Inc., Chicago
Merck & Co., Inc., Rahway, N. J.
Miles Laboratories, Inc., Elkhart, Ind.
Philip Morris & Co. Ltd., Inc., New York
C. V. Mosby Co., St. Louis, Mo.
V. Mueller & Co., Chicago
National Electric Instrument Co., Inc., Elmhurst, L.I., N. Y.
Ohio Chemical & Surgical Equipment Co., Madison, Wis.
Orthopedic Equipment Co., Bourbon, Ind.
Orthopedic Frame Company, Kalamazoo, Mich.
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